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REPORTS

TO THE

LOCAL GOVERNMENT BOARD

ON

PUBLIC HEALTH AND MEDICAL SUBJECTS.

(NEW SERIES No. 77.)

Report to the Local Government Board on Bacterial Food Poisoning and Food Infections, by Dr. W. G. Savage.

[*Food Reports No.* 18.]

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September 1913

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TO THE RIGHT HONOURABLE JOHN BURNS, M.P.
PRESIDENT OF THE LOCAL GOVERNMENT BOARD.

SIR,—

The accompanying report by Dr. W. G. Savage, made by direction of the Board, presents a summary of our present knowledge on the subject of bacterial food infections.

Each year a number of local outbreaks of illness come to the notice of the Board which possess certain common characteristics. They occur most frequently during the warmer months of the year, and are attributable to the consumption of some particular food material, generally of animal origin. The illness usually associated with these outbreaks varies in character and duration, but is not infrequently severe or even fatal. It was formerly thought to be caused by the ingestion of food which contained products of decomposition known as ptomaines, and it is still commonly known as "ptomaine poisoning." Such cases are now generally recognised, however, to result from the presence in the food materials consumed of certain pathogenic organisms, which are either living and capable of active multiplication, or which although killed in the cooking are nevertheless poisonous by means of the toxins contained in them. The term "bacterial food poisoning" is, therefore, more correctly used to describe this disease or group of diseases.

Owing to the sudden onset and rapid course of outbreaks of bacterial food poisoning, they are often practically over before their investigation can be undertaken, and frequently the remains of the food suspected of having been at fault have been destroyed and suitable pathological specimens from those attacked cannot be obtained for bacteriological examination. Largely for these reasons our present knowledge of the etiology of bacterial food poisoning remains in many respects incomplete and unsatisfactory.

The Annual Report of the Medical Officer of the Board for 1890 contained a summary review of outbreaks of food poisoning prepared by Dr. Ballard, which dealt with British cases recorded previous to that date. In the accompanying report Dr. W. G. Savage, who has made a special study of this subject, has continued this review up to the present, and has furnished a history of the most noteworthy recorded cases, both Continental and British, analysed in the light of recent bacteriological knowledge. This conspectus of the subject should prove of value for purposes of reference and comparison in future cases of epidemic food poisoning, and it is hoped that it will assist the progress of further scientific investigation of the subject.

The memorandum issued by the Board to local sanitary authorities in September, 1911, on the investigation of outbreaks of illness suspected to be due to food poisoning is reproduced in Addendum B, p. 68. Attention was there drawn to the scope of the enquiries which should be made in a sanitary district in which cases of suspected food poisoning are brought to the notice of the medical officer of health; enquiries which should be directed both to the

nature and extent of the illness associated with the suspected food, and to the circumstances in which the food may have become infected. Stress was also laid on the importance of provision being made for bacteriological examinations in anticipation of the occurrence of food poisoning, so as to avoid the delay and trouble entailed by special arrangements made in an emergency.

The last paragraph of the memorandum states "The Board are aware that neither medical practitioners nor the general public are under statutory obligation to report cases of food poisoning to the medical officer of health. It is believed, however, that if it is generally understood in a district that the local authority, with a view to safeguarding the public health, is prepared to authorise any necessary inquiries and investigations, the arrangements made will receive general approval and tend to secure the co-operation of all concerned."

For this purpose it is very desirable that the medical officer of health should be in close touch with medical practitioners in his district, and should give them every encouragement to inform him of cases of illness suspected to be due to food poisoning. It should be realised that medical officers of health in this way may be able to connect what appears to be an independent case in one medical practice with similar cases occurring in other practices, and thus be able to trace the illness to a common source.

As Dr. Savage points out, the knowledge which may be obtained from future investigations may have the invaluable result of defining with greater accuracy than is now possible the nature of the precautionary measures which are essential if these outbreaks are to be prevented. Our present knowledge shows the importance of three considerations:—firstly, the association of some outbreaks at least with actual disease of the animals the flesh of which has been eaten; secondly, the probability that in other outbreaks uncontaminated food has received its infection from the tissues or intestinal contents of food animals in which bacterial infection is present (as may happen where a slaughterhouse is used as the place of preparation of such articles as brawn, potted meat, or sausages); and thirdly, the extent to which, when sources of bacterial infection are present, the opportunities of spreading disease are favoured by lack of cleanliness and care in handling, preparing, or storing food. Recent additions to our knowledge, in fact, tend to emphasise rather than to lessen the importance of inspection of meat at slaughterhouses, of the exercise of adequate control in regard to the suitability of places where food is prepared and stored, and of measures to secure cleanliness in all stages of food preparation.

I have the honour to be, Sir,
Your obedient Servant,
ARTHUR NEWSHOLME,
Medical Officer of the Board.

July, 1913.

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SECTION I.

Introductory and Historical.

Outbreaks of disease from the consumption of food have a varied etiology, and at least three groups may be recognised.

A. Those in which the symptoms are due to chemical poisons, such as lead or arsenic, which are not of bacterial origin.

B. Those due to infection of the food by bacteria associated with specific infectious diseases, such, for example, as typhoid fever or tuberculosis.

C. Those due to infection of the food by bacteria which produce toxic substances, either before or after ingestion, to which the symptoms are due.

The present report is only concerned with the third group, and while no single title is accurate and inclusive, the one given—Bacterial Food Poisoning and Food Infections—is sufficiently exact. There is no sharp defining line between this group and the second group, but it may be said that, as a rule, in the third group the toxic features are more in evidence than the infective and that there is but little evidence of case to case infectivity. In actual practice there is usually but little difficulty in defining this group of cases and in distinguishing them from outbreaks of infectious disease spread by food.

Food has been recognised from early ages as a cause of disease, but it is only in modern times that definite information has been obtained as to the nature and origin of the disease-producing properties which may be associated with it. The early conceptions of food poisoning were purely chemical in nature, and this without invoking bacterial action upon the meat to produce the chemical poisons.

In 1820 and 1822, Kerner published his papers upon poisonous sausages, ascribing the poisonous properties to a compound of sebacic acid and a volatile principle. We now know that these outbreaks were cases of botulism and due to a bacillus.

Later conceptions of food poisoning, and particularly of meat poisoning, were chiefly based upon the supposed relationship of the poisonous element to putrefactive decomposition of the food, and very numerous investigations were made along this line of inquiry.

Panum, in 1856, appears to have been the first to demonstrate that the poisonous qualities exhibited by putrid flesh were chemical in nature and not destroyed by boiling. Bergmann and Schmiedeberg, in 1868, obtained a highly toxic substance which they called "sepsin" from putrid yeast and from decomposing blood. After this discovery, other allied toxic bodies were discovered and extensively studied, and the whole group of basic substances with alkaloid characters were called *ptomaines* by Selmi. Selmi, however, did not himself succeed in isolating any of the putrefactive alkaloids, all his experiments being made with extracts, and Nencki, in 1876, was the first to isolate any of these bodies. Brieger (1882 to 1888) isolated and investigated a number of ptomaines.

The ptomaines, being bodies highly toxic to animals (when injected) and being obtained from putrefying meat, were at one time held to

supply the cause and explanation of most, if not of all, cases of food poisoning and illness, and these outbreaks became fairly generally known, especially in this country, as cases of ptomaine poisoning. Indeed, at the present time, not only in popular estimation, but even amongst medical men and in scientific text-books, this term is still largely retained, although, as will be shown later, there is little or no justification for its continued use.

While the ptomaine hypothesis largely held the field as the correct explanation of these outbreaks, another (a bacteriological) conception was gradually being evolved, the two hypotheses developing concurrently and being to some extent complementary to one another.

Bollinger, in 1876, in an important paper read at Dusseldorf, drew attention to the great importance of meat poisoning and to the fact that many outbreaks were associated with pyæmic and septicæmic conditions of the animals from which the food was obtained. He pointed out that the toxins of these diseases were not destroyed by cooking.

In a later paper, in 1880, Bollinger collected the literature of the subject and was able to emphasise further the relationship between human food poisoning outbreaks and septic, pyæmic and gastro-intestinal conditions in the animals whose flesh was consumed. The bacteriological proof of this relationship was not however forthcoming for a number of years.

Amongst the first bacteriological investigations on the subject were those of Klein, in 1880 for the Local Government Board, into the outbreak of food poisoning at Welbeck (Notts), due to eating infected ham. No definite proof was, however, adduced that the bacteria isolated had caused the disease.

The first important landmark in the bacteriological investigation of food poisoning outbreaks was the isolation by Gaertner in 1888 of *Bacillus enteritidis* from a meat poisoning outbreak at Frankenhausen. The bacillus was isolated both from the fatal case and from the organs of the cow, killed on account of enteritis, the consumption of whose meat caused the outbreak.

Since that date this bacillus, or closely allied forms, have been isolated from a very large number of outbreaks both in this country and abroad, and the etiological relationship has been firmly established.

The bacteriology of food poisoning also owes much to Van Ermengem. He investigated a number of outbreaks due to *B. enteritidis* and allied bacilli, while in particular, in 1896 he isolated and studied *B. botulinus*, the cause of an important group of food poisoning outbreaks (botulismus or sausage-poisoning), which were at one time especially numerous in Wurtemburg and other parts of South Germany.

We owe an important further advance in the bacteriological study of food infections to Durham who demonstrated in 1898 that by the use of agglutination tests, the bacilli isolated from food poisoning outbreaks, hitherto all indistinguishable, could be separated into at least two distinct groups. He also drew attention to the diagnostic value of the examination of the sera of patients suffering from food poisoning.

Independently, and nearly at the same time, De Nobele in Belgium came to similar conclusions.

On the epidemiological side a very important summary of the then known etiological facts in relation to food poisoning was written in 1890 by Dr. Ballard for the Local Government Board.

During the last five or six years a fresh impetus has been given to the subject and much additional light has been thrown upon it by the careful investigations which have been made as to the distribution of *B. enteritidis* and other bacilli of the Gaertner group in nature and their relationship to different diseases in man and animals.

Owing to the greater attention which the subject has received, especially during recent years, the number of recorded outbreaks of food poisoning is now considerable. Ostertag, in 1902, was able to collect records of 85 epidemics in the period 1880–1900, mostly from German sources. Apart from Ballard's report which dealt with 14 groups of cases, there has been no systematic analysis of outbreaks in Great Britain. In the present report the records of 79 outbreaks in Great Britain and Ireland have been collected and studied. Some of them have been described in detail, but for others the available information is very meagre. In addition the facts and investigations recorded in regard to a number of continental outbreaks have also been utilized.

SECTION II.

Certain Special Kinds of Food Poisoning.

In general, food poisoning outbreaks are more conveniently grouped and studied according to their bacterial origin rather than in relation to the kind of food infected. Certain kinds of food poisoning are, however, on account of their special nature, more conveniently studied from the food standpoint. These will be briefly considered in the present section.

Mussel Poisoning.

Epidemics of typhoid fever have been frequently traced to the consumption of mussels as well as to other shellfish. In addition to these epidemics outbreaks of acute poisoning attributed to the consumption of mussels have been recorded on numerous occasions.

In the recorded outbreaks the onset has usually been rapid and within a few hours of consuming the mussels, but occasionally it has been delayed for even as long as 18 to 24 hours. While the ordinary food poisoning symptoms are met with to some extent, *i.e.*, vomiting, diarrhœa and epigastric pain, they may be absent, and only very rarely are dominant symptoms. The main symptoms are referable to the nervous system. In the Leith outbreak, for example, Combes, who described it, gives the chief symptoms as—prickly feeling in the hands and feet, feeling of constriction of the mouth and throat, difficulty in swallowing and speaking freely, numbness about the mouth, gradually extending to the arms, and muscular weakness of the limbs.

In the Dublin cases, described by Sir Charles Cameron, the chief symptoms were vomiting, dyspnoea, swelling of the face, want of co-ordination of movement, spasms, especially of the arms. The

onset started with prickly pains in the hands. The symptoms are often very severe and not infrequently fatal, as shown in the following particulars of a few outbreaks in the British Isles :—

Place.	Date.	Number of Cases.	Number of Deaths.
Leith	1827	30	2
Ramsgate	1880	2	0
Liverpool	1888	3	0
Dublin	1890	7	5
Barry	1909	19	1
		61	8

Case mortality, 13 per cent.

Many explanations have been advanced to account for the causation of mussel poisoning. These are carefully reviewed by the late Dr. Bulstrode in his report (1894–95) to the Local Government Board on "Oyster Culture in relation to Disease." The earlier views ascribing the pathogenic properties to copper poisoning, starfish spawn, mussels eaten during the spawning season, mussels eaten in a stale or dead condition may be dismissed as untrue or inadequate as explanations generally applicable although metallic poisoning cannot be excluded as a possible cause of illness in exceptional cases or even in small outbreaks.

The Wilhelmshaven cases directed attention into another field. From a number of the poisonous mussels Brieger obtained a poisonous base, which he regarded as a ptomaine, and called *mytilotoxin*. The symptoms produced in animals by the injection of this substance are, according to Brieger, similar to those produced in mussel poisoning. When mussels have caused illness it has invariably been found that they were taken from positions liable to sewage pollution, such as from dock walls or vessels in harbours. Although in opposition to this there is the undoubted fact that very large numbers of this shellfish, taken from sources equally exposed to sewage pollution, are eaten without harm, it is highly probable that their toxic properties are derived from their polluted surroundings. Schmidtmann, for example, found that healthy non-poisonous mussels placed in polluted water from which poisonous mussels were obtained themselves became poisonous, while the harmful mussels kept in clear running sea-water became non-toxic.

No modern investigations appear to have been made upon the bacteria causing poisonous properties in mussels, but McWeeney ([72]),* in 1890, published some preliminary investigations and quotes Lustig as having, in 1888, isolated from the livers of specimens of mussels a bacillus rapidly pathogenic to rabbits and guinea pigs.

The most probable view is that mussels become poisonous from the production in them of chemical poisons elaborated by the vital activity of bacteria derived from their sewage contaminated surroundings, but we are ignorant of the special bacteria concerned, the conditions which cause the production of these poisons, and whether the toxic properties are due to mytilotoxin or to some other bacterial poisons.

* The small figures in brackets refer to the bibliography on pp. 72–80.

Cheese poisoning.

Outbreaks of sudden illness from the consumption of cheese are not very uncommon, cases being more frequently recorded in the United States.

The chief symptoms consist of diarrhœa, vomiting and abdominal pain. There is usually some degree of fever; rigors may be met with and symptoms of collapse are often present. The symptoms appear to vary greatly in severity, sometimes being very severe; no deaths were however recorded in either the Michigan (1883–84) or London (1901) outbreaks.

The Michigan outbreak consisted of about 300 cases caused by the consumption of 12 different cheeses. It is not clear if they were obtained from the same source. The incubation period was mostly 2–4 hours. In London a considerable outbreak occurred in 1901, the cases being rather scattered: 17 were traced in Finsbury, 21 in Bermondsey, and others in different parts. The symptoms came on in from two to eight hours after eating the cheese which was "Dutch Cheese," and all made by one firm in Holland. In this outbreak, as in others, the cheese was normal as regards taste, odour and appearance.

The first scientific contribution upon the causation of cheese poisoning was made by Vaughan in connection with the 1883–84 Michigan outbreak. From the aqueous extract of the cheese specimens Vaughan separated a crystalline poisonous body which he called *tyro-toxicon*. Tyro-toxicon is not an alkaloid, but in its chemical reactions, and to some extent in its physiological action, resembles diazobenzene.

This substance has since been isolated on a number of occasions from cheese which has caused outbreaks of poisoning. In the London outbreak, mentioned above, the analyst who examined the cheese reported that he obtained a small amount of a body possessing the chief characteristics of tyro-toxicon. Later investigations have shown that tyro-toxicon is not present in all poisonous cheeses, and that in such cases at least the harmful properties must be ascribed to other causes.

Bacteriological investigations have also been carried out. In 1895 Vaughan and Perkins ([143]) described an outbreak of 12 cases from eating cheese. From a piece of the cheese they isolated two bacilli, one of which elaborated an active toxin. This bacillus was a strain of *B. coli communis* or *B. lactis aerogenes*, chiefly differing from the former in its more rapid milk clotting power and its failure to produce indol. Both filtered and heated cultures rapidly killed laboratory animals. The toxins were partially heat resistant, not being entirely destroyed by 14 minutes heating to 100° C.

Owing to a mistake in labelling, the toxicity of a sterilized culture was demonstrated upon a human subject who was made very seriously ill by the injection of ten drops of a concentrated (50 per cent.) germ free milk culture, the symptoms commencing within half an hour.

In subsequent investigations by Vaughan, organisms of the colon group were isolated which produced powerful toxins which were also heat resisting.

Holst ([54]) records five different outbreaks of cheese poisoning observed by him within a period of five months. He isolated *B. coli communis* from each of the five lots of cheese—(a Norwegian cheese called "Knet Käse"). He carried out some interesting feeding experiments with quite young calves. When calves were fed with *B. coli* from ordinary sources and from non-poisonous cheese, no diarrhœa or other symptoms were caused, but when calves were fed with the *B. coli* from the cheese—four strains from four separate outbreaks being used in individual experiments—the calves suffered from diarrhœa and in some cases died. In the latter case the bacilli were recovered postmortem from the spleen, blood, &c. The bacilli from the poisonous cheese were also highly virulent to rabbits.

It would appear to be probable that cheese poisoning, like other forms of food poisoning, is usually due to bacteria which produce powerful toxins. The bacterial investigations just recorded suggest that specially virulent strains of *B. coli* are concerned, but further investigation is desirable. Gaertner group bacilli have not up to the present been found in poisonous cheese.

The bacteriological investigations in the London outbreak showed the presence of *B. coli communis*, *Bacterium lactis* and other bacteria, but no bacteriological relationship to the outbreak was disclosed.

Potato Poisoning.

A considerable number of outbreaks of food poisoning have been reported from the consumption of potatoes. It is rather noticeable that many of these outbreaks have occurred amongst troops.

At one time they were ascribed to solanin poisoning, but later investigations have shown that the amount of solanin is quite insufficient to produce the symptoms. Later investigations have shown that the poisoning properties are due to bacterial activity.

At the present day there are no sufficient grounds for separately classifying potato poisoning outbreaks as different from those due to the consumption of meat and other kinds of food. The symptoms, for example, are those of ordinary food poisoning and not distinctive as in mussel poisoning.

Their comparative frequence is no doubt due to the fact that peeled potatoes are valuable nutrient media for bacteria. In my series, one of the outbreaks (No. 65) is almost certainly an example of potato poisoning, although the recorder of the outbreak does not ascribe this as the cause and the investigations which were made were on other lines.

This outbreak is described and further facts in regard to potato poisoning are considered in Section VI.

Ice-cream Poisoning.

Outbreaks of poisoning from eating ice-cream are fairly frequent but, even making allowances for the numerous unrecorded outbreaks, less so than might be anticipated from the commonly unsatisfactory conditions under which this food material is prepared.

Although frequently grouped as a special class of food poisoning, ice-cream poisoning outbreaks do not differ in any essential particular from those due to the consumption of other foods, and the facts may be conveniently considered with the other data in the following sections. In the British series included in this report six outbreaks have been ascribed to the consumption of ice-cream.

SECTION III.

The Clinical and General Features of Recorded Outbreaks.

The facts set out in this section are derived from a study of the details of the British outbreaks summarised in Table I., together with particulars obtained from many of the Continental outbreaks (Table II.). (*See* Addendum A, p. 61.)

These facts are more particularly considered from the point of view of the light they throw upon the etiology of food poisoning and food infections.

In this section the peculiar symptoms and other characteristic features of the group of cases due to infection with *B. botulinus* and known as botulism are not considered. They are more conveniently grouped together under botulism in Section IV.

Incubation Period.—The average incubation periods in the individual recorded outbreaks are set out in Tables I. and II. These figures show that the period between consumption of the food and the onset of symptoms varied greatly, being in some cases as short as half an hour and in others as long as 30 hours or even longer. The incubation period most frequently noted was from 6 to 12 hours. Also not only does the incubation period vary in different outbreaks, but in the same outbreak widely different incubation periods have been recorded. For example, in the Derby outbreak of 1902 (No. 30) the following was recorded :—

In	3	instances	the	incubation	period	was	...	4 hours.
„	8	„	„	„			between	4 and 8 hours.
„	18	„	„	„			„	9 „ 12 „
„	6	„	„	„			„	13 „ 15 „
„	15	„	„	„			„	16 „ 20 „
„	4	„	„	„			„	21 „ 24 „

These variations are easily explained. The symptoms are the manifestation of the action of the toxins of the Gaertner or other bacilli concerned upon the tissues. They are due either to the toxins pre-formed in the food outside the body, to the toxins produced in the body after ingestion of the food, or usually to a mixture of both.

In outbreaks due to toxins alone, no living bacilli being present, the symptoms are rapidly manifested. For example, in the 1906 Partick outbreak (No. 34) in which Gaertner toxins only were ingested with the tinned beef the onset of symptoms was mostly within 1 to 2 hours and in all within 4 hours. In the Wigan 1911 outbreak (No. 78) with a similar origin all the eleven victims were severely ill within 2 hours of eating the beef.

The majority of outbreaks must be looked upon as being caused by a mixture of bacilli and toxins, the bacilli having had time to grow in the food before it was consumed. In such cases varying incubation periods are likely to be met with since the amount of toxin ingested will vary with the quantity of food eaten and the degree of local bacterial contamination of the food. In a broad general way these factors do bear some relationship to the length of incubation period and the severity of the symptoms.

In general the longer the period from the initial contamination to the consumption of the food the shorter the incubation period. This was demonstated in both the Chesterfield and Wrexham outbreaks. On the other hand, in the Mansfield outbreak (No. 17) the converse was the case, and, speaking broadly, those persons who ate the potted meat soonest after its manufacture had a shorter period incubation, while those who ate it on later days had a later incubation period.

Symptoms.—As a rule no symptoms are present during the incubation period. The onset is usually sudden. The symptoms are essentially those of marked gastro-intestinal irritation but are frequently accompanied or followed by symptoms pointing to involvment of the nervous system.

Three symptoms—vomiting, diarrhœa and abdominal pain—are met with in almost every outbreak and frequently in every case. Vomiting is the least characteristic of these symptoms and is not invariably present. In the Chesterfield outbreak, for example, it was present in 75 per cent. of the cases.

The diarrhœa is usually very severe with repeated actions which, as a rule, are extremely offensive. Later in the attack the actions are more watery and frequently green in colour.

The abdominal pain is nearly always severe ; in some outbreaks it is spoken of as agonising, and frequently is the first symptom complained of. Pain in other regions, such as the back or limbs, is not uncommon.

Prostration, marked and often persisting long into convalescence, is usually present and is a characteristic feature of most of the severe attacks. Marked collapse occurs in severe cases, often giving rise to great anxiety. Cold sweats and even rigors are mentioned as symptoms in some outbreaks.

As a rule a moderate elevation of temperature is present in the early stages of the illness. The tongue is heavily coated and the breath offensive. Headache is usually present and giddiness is frequently recorded as a symptom.

Numbness and cramp in the limbs are less commonly mentioned. Severe cramps were prominent in the Chester and also in the Wigan outbreaks.

Of other conditions which may be present the most interesting are herpes and other rashes. Herpes was recorded in a number of cases in the Bacup, Wrexham and Derby outbreaks, and rashes were specially mentioned in the Derby, Ashton-under-Lyne, Warrington and Tollesbury outbreaks. The rash is usually stated to be erythematous or urticarial in character and may be followed, as was noted in the Derby outbreak, by well-marked desquamation.

The severity of the symptoms varies very greatly in different outbreaks, and even in the same outbreak.

The duration of the symptoms shows considerable variation, but they usually diminish after two or three days although marked prostration may persist much longer. The Chesterfield outbreak is in this way typical: "most of the acute symptoms disappeared in 24 to 48 hours, but some patients were left in a very weak state and were several weeks before they recovered their usual state of health."

In addition to the ordinary cases with symptoms of acute gastro-enteritis cases are occasionally met with which show a rather different clinical picture. The acute symptoms are less severe while the patients are ill for several weeks. The clinical course of their disease, apart from the acute onset, closely resembles typhoid or paratyphoid fever. The best English examples occurred in the Mansfield, Murrow and Chesterfield outbreaks, one case in each, the other cases showing the usual symptoms.

In the Mansfield outbreak a boy aged 14 had an unusually protracted illness. He had no diarrhœa and did not vomit. He was very ill from the first and maintained a considerable temperature for several weeks with typhoid-like symptoms. He complained of abdominal pain and headache. He gradually improved after the fourth week.

In the Murrow case the woman only received a small dose of toxin and Gaertner group bacilli; she became ill with diarrhœa and vomiting within 24 hours but subsequently the symptoms more nearly resembled those of enteric fever, the patient dying 20 days after the onset.

In the Chesterfield case the patient, a boy aged five, was very drowsy for two days after eating the pie, but was only sick once or twice and not purged more than two or three times. He had a high temperature for a fortnight or so, but otherwise had few symptoms. He gradually recovered.

Similar cases have been recorded in Continental outbreaks. In the Gaustad (No. 83) outbreak one of the sufferers died after 27 days of illness, while in an outbreak in 1906 described by Prigge and Sacks-Müke, in which 16 persons were ill after eating roast pork, one case ran a typhoid-like course.

These cases are of special interest in their clinical similarity to paratyphoid fever, and the possible relationship between this disease and food-poisoning.

Case to Case Infectivity.—In the records of outbreaks it is extremely rare to meet with cases of secondary infection. In the present British series the only recorded instance is in the Wrexham outbreak. One woman died from infection from the pork-pie, and her son (age 28), who lived with her, did not partake of any pie, but was taken ill three days after her death with exactly the same symptoms and died 15 days later. Other members of the family—notably a sister and two daughters of the deceased woman—suffered from the same symptoms. "No pies were eaten by these women, but they nursed the deceased woman and son." "An examination of their blood proved them to have suffered from the same complaint as the deceased persons."

A few cases are recorded in foreign literature. In the Frankenhausen outbreak (No. 80) the mother of the fatal case was attacked with the same symptoms, although she had not eaten any of the incriminated food. It is probable that case to case infectivity is potentially as great as in typhoid fever, but the cases being for the most part very acute and quickly ending, there is but little opportunity for secondary infection.

Age and Sex Distribution.—There is no evidence of any special incidence on either sex or at any age period. The age and sex distribution in any outbreak seems to depend entirely upon the accidental age and sex distribution of those who ate the food.

Infectivity Rate.—As a rule the infectivity rate is high. Not infrequently all who ate the infected food became ill (100 per cent.); in many outbreaks it is recorded as 80 per cent. or more, and in only a comparatively few of the records is it given as under 50 per cent. The figures are given in Table I. as far as they are available.

I am inclined to believe that the infectivity rate is not nearly as high as the table shows it to be. Unless special inquiry is made and all the food traced, attention is apt to be concentrated upon the cases of illness and the families and individuals overlooked who consumed the food without harm. In a number of instances, for example, it is recorded that those who ate the food on the day it was prepared were not affected or were affected to a lesser extent. These cases should all be taken into consideration when the infectivity rate is being calculated. Specific instances are given on page 47.

Probably the form in which the food is taken, and especially if alone or with other food, is important. Undoubtedly individual idiosyncrasy plays a part. As instances of this the following may be mentioned. In the Mossley outbreak (No. 49) two women divided a meat-pie between them, each eating half. One woman was only slightly affected, and recovered within two days; the other commenced to be ill after four hours, suffered severely, and died after nine days' illness.

In the Longton cases (No. 41), one person ate the salmon to which the illness was ascribed on three different occasions and had more of it than anyone else, yet she had a comparatively mild attack, while her husband, who consumed much less, was very ill and died.

In the Warwick outbreak (No. 59) the grocer who sold the boiled beef was said to have had four meals from it without ill effects, although, as the bacteriological results showed, the meat was infected with living Gaertner bacilli.

Case-Mortality Rate.—The case-mortality rate or fatality varies greatly in different outbreaks. In a considerable number, although the symptoms were severe, no deaths resulted. The number of deaths are given in Table I. for British outbreaks, and in Table II. for Continental. Dealing with all the British outbreaks mentioned in the Table, as far as the figures are available, there have been some 3,098 cases with 67 deaths, a case-mortality rate of 2·16. If the outbreaks in which true Gaertner group bacilli have been definitely, or at least probably, shown to be the cause are separately considered from those in which the bacteriology has not been definitely worked out, there have been 40 deaths; a case-mortality rate of 2·7. The

outbreaks definitely due to Gaertner group bacilli have shown a very variable case-mortality rate ; thus, in the Tollesbury outbreak there were no deaths out of 80 cases, at Chesterfield and Derby the case-mortality rate was less than 1 per cent., at Wrexham it was 4·7, at Limerick 12, and at Murrow 17 per cent.

The virulence of the particular strain of bacilli concerned, the time which it has had to grow upon the incriminated food before consumption, and the temperature of growth, are the factors which probably more especially control the case-mortality rate.

Pathological Appearances.—In severe cases the gastro-intestinal symptoms are often very intense, and it might be anticipated that very definite pathological changes would be shown in the gastro-intestinal tract in fatal cases. The changes found on post-mortem examination are, however, often slight compared with the severity of the symptoms. The mucous membrane of stomach and intestine is swollen and congested and shows minute hæmorrhagic erosions, but often nothing else. The kidneys usually show cloudy swelling, the spleen and liver are congested. These may be the only pathological appearances.

The following concisely describes the findings in a case at Wrexham, fatal after thirteen days' illness :—

> "The stomach was generally congested, but the congestion was more marked towards the pylorus, and several extravasations were noticed. The small intestines were much inflamed, many small submucous extravasations of blood had taken place, and many patches of slight superficial ulceration were visible. The large intestine was also congested, especially the rectum. The heart was normal. The liver, kidneys and spleen were congested."

In the Limerick cases, Professor McWeeney found that the mucous membrane of stomach and intestines showed patches of white, prominent granules, each as large as a pin's head and consisting of nodules of tumid lymphoid tissue. The liver also showed little, indistinct pale-yellowish areas or spots, which microscopic examination showed to be due to intense fatty degeneration of the liver cells at these places. These areas are evidently similar to the necrotic areas met with in the liver, &c., of animals naturally or artificially infected with Gaertner group bacilli.

Seasonal Prevalence.—The influence of the time of year is very marked, food poisoning outbreaks being far more prevalent in the hotter months. This is shown in Charts 1 and 2, Chart 1 giving British outbreaks and Chart 2 a number of continental outbreaks in addition. (*See* Addendum D, p. 81.)

This is true of outbreaks selected without regard to etiology, and it applies equally when those outbreaks alone are considered which were shown to be due, probably or definitely, to one or other member of the Gaertner group (Chart 3). It will be noticed that the months of greatest prevalence in Charts 2 and 3 are not identical, although there is a general agreement. The number of outbreaks is, however, too few to draw deductions from this fact as to a different etiology in the outbreaks shown to be due to Gaertner group bacilli and those not worked out, or ascribed to other bacilli. Indeed, as shown in Chart 3, the British and Continental outbreaks do not show the same monthly prevalence.

Physical Appearance of the Incriminated Food.—As a general rule, in the outbreaks studied the food affected was not noticeably altered in either appearance, taste or smell. This is true both of cases in which the bacteriology was obscure or not investigated, and of outbreaks from which Gaertner group bacilli were isolated.

In the days when food poisoning outbreaks were only beginning to be scientifically investigated, the innocuous and normal appearance of poisonous food was imperfectly recognised. A classical illustration is furnished by the Ghent outbreak in 1895 investigated by Van Ermengem. In this outbreak, the slaughter-house inspector (a veterinary surgeon) was so certain that the suspected meat (saveloy) in the absence of any abnormal signs, could have no connection with the outbreak that he ate two or three pieces of it, to demonstrate its harmlessness. He was attacked with severe cholera like symptoms and died five days later, the Gaertner bacillus being recovered post-mortem.

Sausages and flesh inoculated with Gaertner group bacilli show no change in appearance or odour even after several days' incubation. Inoculated brawn also appears unaltered to superficial inspection, but the bacterial growth in the otherwise clear jelly is noticeable to a trained eye. Thus Delépine in the Accrington (No. 33) outbreak noticed that one of the surfaces of the sample was smooth and covered with a thin layer of jelly, and that the jelly was slightly turbid, due to very numerous colonies of bacteria.

In the recorded outbreaks it is noteworthy that in several instances of poisonous tinned meats the physical appearances of the meat were not perfectly normal. The following may be mentioned.

Sheffield (No. 24), due to tinned corned beef. "Nothing was noticed about the meat except that it was less solid than usual and that the jelly was rather oily. It had no smell, and although a few, it would seem, noticed some nasty flavour about it, the majority of the purchasers observed only an unusual saltness."

Partick (No. 34). In most cases no complaints were recorded, but in one case two men who ate the meat declared that it was bad as soon as they had tasted it. Dr. Brown, who recorded the outbreak, examined a portion of the meat in one of the houses and states that "a somewhat heavy odour was perceptible, and it was noted that the jelly of the meat instead of being firm was soft and liquefied. No evidence of putrefaction as judged by the sense of smell was present."

St. Helens (No. 60). The corned beef in the affected tin "was noticed to be softer and moister than usual, which was blamed on the weather. There was no unpleasant smell about the meat, and the persons who bought it appear only to have noticed its softer and moister condition, although one or two of the sufferers stated that while eating it they noticed that it had a peculiar taste and that they thought it was not good."

Astley (No. 61). When the tin of tongue was opened one of the persons who ate it declared that "it was watery and smelled." It had been on the shopkeeper's premises for eight months.

Chester (No. 71). The shopkeeper when he opened the tin of corned beef noticed that the meat was moist and refused to sell any. The next morning he smelt it, found it sweet, cut the top part off

(which he gave away) and sold the rest. When the Medical Officer examined the remainder in the shop it appeared normal and had no smell.

These outbreaks can all be assigned, definitely or with strong probability, to Gaertner group toxins or bacilli, and they suggest that in cases in which these bacilli or their toxins are left in the tins through infection and subsequent faulty sterilization physical changes in the meat are likely to occur which, although slight and ill-defined, should be demonstrable by the trained observer.

In the three tinned salmon outbreaks no complaints as to the physical condition of the salmon were made. If present, however, any abnormalities would probably be masked by the strong flavour and colour of the salmon.

Nature of the food acting as the vehicle of infection.—Dealing with the British outbreaks recorded in Table I in one (No. 8), particulars are not given in regard to the food eaten or the animal from which derived. In 12 of the remaining 78 outbreaks the vehicle of infection was a non-flesh food, *i.e.*, ice cream, 6 ; cream, 1 ; milk, 3 ; potato (probably), 1 ; pineapple jelly, 1.

The remaining 66 were all due to flesh foods as follows:—

Pig.—Brawn	7		
Pork pies	6		
Brawn + pork pies	1		
Pork or ham	5		
„ pies + salt pork	1		
Tinned tongue	1		
Sausage rolls	1		
Pork + brawn	1		
Chitterlings	1		
		24	
Ox or Cow.—Brawn	2		
Beef pies	2		
Flesh	6		
Tinned beef	6		
Fresh tongue	1		
Pickled tongue	1		
Udder	1		
Steak and kidney pie	1		
		20	
Pig and Ox.—Potted meat	1		
„ tongue	1		
„ meat and pork pie ...	1		
Ham and beef	1		
		4	
Rabbit		1	
Calf (all veal pies)		3	
Goose		1	
Shrimps		1	
Hough and potted chicken		1	
Carried forward		55	

Brought forward			55
Fish.—Tinned salmon	4		
Fish	1		
Kippers	1		
			6
Animal unnamed.—Tinned tongue	2		
Meat pies	1		
Frozen meat	1		
Sausage skins	1		
			5
			66

Three of the 44 continental outbreaks of Table II were due to the consumption of other than meat foods, *i.e.*, tinned beans (1); pudding (2). The remaining 41 were distributed as follows :—

Pig.—Pork or ham	4		
Chopped meat	2		
Sausages (blood, liver, &c.)	3		
Salt pork + schwartenmagen	1		
			10
Ox or Cow.—Flesh	10		
Chopped meat	1		
Sülze	1		
			12
Pig and Ox.—Flesh	1		
Pork + sausages, &c.	2		
			3
Horse.—Flesh	2		
Chopped meat	1		
Sausages + flesh	2		
			5
Calf.—Veal	3		
Sausages	1		
Flesh + internal organs...	1		
			5
Sheep.—Leg of mutton			1
Fish			2
Animal unnamed.—Sausages	2		
Chopped meat + sülze ...	1		
			3
			41

These figures show that in nearly all the British outbreaks the meat was derived from pigs or cattle, while in the continental cases the horse was also concerned. The almost complete absence of the sheep from the list is striking, only one instance being recorded, although it should be mentioned that in one English outbreak (Surbiton No. 22) some ribs of lamb appear to have been also secondarily affected. Excluding the cases in which the animal is not mentioned and dealing

only with meat foods the following figures and percentages are obtained :—

Animal.	Outbreaks.			Percentages.		
	British.	Continental.	All.	British.	Continental.	All.
Pig	24	10	34	39	26	34
Ox (cow) ...	20	12	32	33	32	32
Pig + ox ...	4	3	7	6·5	8	7
Calf	3	5	8	5	13	8
Horse	0	5	5	0	13	5
Fish	6	2	8	10	5	8
Other animals...	4	1	5	6·5	3	5
Total ...	61	38	99	—	—	—

The nature of the food which carried the infection is of equal interest.

The British outbreaks show the following :—

Brawn (in one case with roast pork)	10
Meat pies (in several cases with other meat) ...	14
Brawn + meat pies	1
Flesh (different forms but not tinned)	17
Tinned meat	9
,, fish	4
Sausages	1
Potted meat (apart from brawn)	4
Other forms	6
	66

A certain number (about 35 per cent.) of the cases were due to the consumption of fresh and not made up meat but in the great majority the vehicle of infection was some form of prepared meat, either simply tinned (20 per cent.) or usually specially prepared (about 45 per cent.). In no less than 38 per cent. of the cases brawn or meat pies was the vehicle of infection.

The continental outbreaks show a much higher percentage of outbreaks due to eating the flesh itself, not infrequently raw or in the form of chopped meat, while in a number of instances some form of sausage was the vehicle of infection.

SECTION IV.

BACTERIA CONCERNED IN FOOD POISONING AND FOOD INFECTIONS.

The bacteria concerned in food poisoning and infections can be included in three groups, the classification being based on convenience rather than on scientific considerations, *i.e.* ;

a. The Gaertner group of bacilli.

b. Non-Gaertner aërobic bacilli—such as *B. proteus*, *B. coli*.

c. *B. botulinus*.

The subject of the present section will be dealt with under the four following sub-sections :—

A. Findings in the recorded outbreaks.
B. Relation to food poisoning of *B. coli* and allied intestinal organisms.
C. Ptomaine poisoning and the putrefactive bacteria.
D. Botulism.

The Gaertner group itself is considered in a separate section (Section V).

A. Findings in Recorded Outbreaks.

Dealing with the British outbreaks first, the chief findings are mentioned in Table I. In about half of them no bacteriological investigations at all were made, sometimes because there was no material left to examine, occasionally because chemical examinations alone were undertaken, but usually because no full investigation was made into the outbreak, which might have enabled its source and nature to be ascertained.

Of the British outbreaks the first twelve were described by Dr. Ballard in his report. While many of them were bacteriologically examined, the examinations were all made before 1890 when bacteriological technique was not well advanced and when insufficient information was available as to the bacteriology of normal meat, &c. The bacteriological investigations made in these outbreaks are sufficiently described in previous Reports to the Local Government Board.

Excluding investigations made before 1890 and 26 outbreaks in which no bacteriological investigations were made, in the remaining 38 outbreaks some bacteriological examinations were undertaken. The bacteriological findings in these 38 cases were as follows :—

1. Bacilli of the Gaertner group isolated or shown by agglutination tests tc be the cause ... 27 outbreaks.
2. Other organisms found to be present ... 11 „

In 71 per cent. of the investigated cases Gaertner group bacilli were isolated or shown to be the cause.

The bacteriological findings in the eleven cases associated with organisms other than those of the Gaertner group may be briefly described. Two of them were cases of ice-cream poisoning. In both *B. coli* (in one case pathogenic *B. coli*) were isolated. No special examination for Gaertner group bacilli appears to have been made and no sera were tested.

One was a case of poisoning from milk. Several samples of the milk were examined, but only after the lapse of three days from the time of the consumption of the milk. From one of the milk samples a *B. coli* strain was isolated. pathogenic to guinea-pigs. This strain was partly agglutinated by the blood of one patient and to some extent by the blood of another patient. These sera gave no reaction with *B. enteritidis* or with the strain isolated from the Derby outbreak, but were not apparently tested with other varieties of the Gaertner group. Professor Delépine, who conducted the examinations, failed to find *B. enteritidis*. He remarks : " It is quite possible that a *B. enteritidis* was also present, but owing to the extraordinary number of bacteria which were present in the milk when it reached me I may have been unable to detect it."

One of the outbreaks (No. 57) was that reported as being due to the use of contaminated sausage skins. The bacteriological examination of some of the same batch of skins was inconclusive. The skins had a disagreeable smell, and the whole history of the cases is different from ordinary cases of Gaertner poisoning.

Outbreak No. 65 is a further case. In this case the only food examined was the oil. This was examined chemically and bacteriologically. The chemical analysis did not disclose any substance which could have been the cause of the illness, but the inoculation experiments showed the presence of some toxic substance causing peritonitis in rabbits. Since, however, the cause of the outbreak was almost certainly due to infected potatoes, and these were not examined, it may be claimed that no proper bacteriological investigation took place in connection with this outbreak. Indeed, as will be mentioned subsequently, this was probably an outbreak due to Gaertner toxins.

In the remaining six outbreaks the food eaten was some form of meat.

The bacteriological results obtained were the following :—

No. 17. Potted Meat.—Two samples were examined by Dr. Klein, but were only received 11 and 15 days respectively after the potted meat had been manufactured. As might be anticipated, a large number of organisms were present, especially *Proteus vulgaris* and *B. coli.* Mice fed with the food showed no ill effects, but the Proteus strains caused death in guinea-pigs by subcutaneous injection, being, if anything, more virulent than when isolated from ordinary sources. Apart from their presence in the meat and the possibly heightened virulence of the Proteus isolated, there was nothing to connect these organisms with the outbreak.

No. 55. Steak and Kidney Pie.—Although *B. coli* were present in large numbers, careful examination failed to find Gaertner group bacilli and the sera of four patients failed to agglutinate any of the Gaertner group strains or the *B. coli* isolated from the pie. Anaërobic spore-bearing bacilli were absent and there was no evidence as to the bacterial cause of the outbreak.

No. 60. Tinned Meat.—Bacteriological examination of the meat failed to showed any Gaertner or coli group bacilli. A number of specimens of blood from patients were tested against several strains of Gaertner bacilli and two strains of *B. coli.* The two coli strains were agglutinated to some extent but none of the other organisms. An anaërobic bacillus resembling *B. botulinus* was isolated from the tinned meat, and this was agglutinated weakly by the sera of patients.

No. 67. Beef and Ham.—Specimens of the beef and ham and of the broth in which they were cooked were bacteriologically examined but were only received four days after the poisoning took place. It is not, therefore, surprising that the bacteriological results were inconclusive. *B. coli* and bacilli somewhat resembling true Gaertner group bacilli were isolated but there were no facts to connect them etiologically with the outbreak. No serum tests were made.

No. 72. Brawn.—The brawn was bacteriologically examined but no

Gaertner group bacilli were isolated. *B. coli* were present and several other kinds of bacilli but none could be shown to be concerned in the outbreak. Agglutination tests with the sera of 14 patients gave no clear reactions, although bacilli isolated from the brawn and bacilli associated with food poisoning outbreaks were employed. The examination of the fæces of nine patients gave indefinite results.

No. 79. Roast Pork.—Careful bacteriological examinations were made but the report does not give definite information. Apart from *B. coli*, a bacillus (Bacillus I.) was isolated from the outside of the meat which agglutinated the sera of two patients. This bacillus was only slightly pathogenic and it is not clear whether it was a *B. coli* or a Gaertner group organism.

The bacteriological examinations of these British outbreaks indicate that while Gaertner group bacilli have been shown with reasonable probability to be the cause of most outbreaks they have not been found in every case. If the outbreaks due to the consumption of some form of meat food alone are considered it will be found that out of 32 outbreaks bacteriologically examined one or other variety of Gaertner group bacilli was shown to be the cause in 25 cases, a percentage of 78.

It will also be noticed that in all 11 outbreaks bacteriologically examined without finding Gaertner group bacilli there were no deaths. Put the other way, in every outbreak severe enough to cause deaths bacteriological examinations, when made, showed the presence of Gaertner group bacilli or their toxins.

While this may be advanced by some as showing that the less severe outbreaks are not always due to Gaertner group bacilli it should be remembered that the true bacterial cause of any outbreak of food poisoning is far more likely to be ascertained if the organs of fatal cases are available for examination. The failure to find Gaertner bacilli in cases in which there were no deaths may, in at least many outbreaks, be ascribed to the unsatisfactory nature of the material available for examination. The bacteriological examination of the incriminated food itself may be difficult and unsatisfactory, in part due to delay in transmission to the bacteriologist, in part to the growth of other organisms which may obscure or even totally eliminate the bacillus responsible for the outbreak, and in part to the possibility that the food submitted for examination is either not part of that incriminated or that the particular portion selected for examination is free from the bacilli concerned in the outbreak.

For example, in outbreak No. 55 the steak and kidney pie examined was one of a batch of four, two only of which had been eaten with harmful results. No part of the pie examined had been eaten, and it may possibly have been free from the bacilli which were evidently present in the other two, the consumption of which caused illness.

In contrast to the difficulties met with in the examination of the food itself is the ease with which the bacteriological examination of the internal organs of fatal cases yields Gaertner group or other bacilli which may be present. Such bacilli are usually very abundant and present in pure culture.

In every one of the 44 Continental outbreaks one or other member of the Gaertner group of bacilli was isolated. While this is very significant the data are not so reliable for statistical purposes as the British outbreaks recorded above, since only the more completely worked out outbreaks are likely to be reported in the German medical press or other sources available to me. The British outbreaks on the other hand have not in any way been selected, but all outbreaks have been included of which I had any information, whether complete or not.

B. proteus or other putrefactive bacilli (apart from *B. coli*) were only isolated in one outbreak, No. 17. The possibility of food poisoning of putrefactive origin is considered in Sub-section C.

B. Relation to Food Poisoning of B. coli and Allied Intestinal Organisms.

This is a convenient place to consider how far *B. coli* and allied intestinal bacilli, other than true Gaertner group organisms, can set up food poisoning outbreaks and cases.

Inasmuch as the *B. coli* group (using the expression in a comprehensive sense to include *B. coli communis* and a number of allied forms) is a group of organisms of very varying virulence which may be highly pathogenic to man, it is quite a possible supposition.

In none of the outbreaks included in the tables could this group of organisms be claimed as the cause. They were present in the food stuffs examined, as set out above, but no etiological connection was established. Their being present is of no significance since they are usually present in identical but non-poisonous foodstuffs (*e.g.*, ice-cream).

Fischer ascribed an outbreak at Grünthal in 1895 to a variety of *B. coli* which produced a toxin that withstood 10 minutes at 80° C. Ten persons ate "Leberpastete." All fell ill about 18 hours after with fever and gastro-enteritis, but rapidly recovered. A coli like bacillus (*B. Grünthal*) was isolated from the food. Mice fed with the paste died after several days. They showed *post-mortem* a hæmorrhagic enteritis with grey white masses in the liver and necrotic areas in the liver and spleen, the special bacillus being present in these areas. Apparently the bacillus was not isolated from the excreta, etc., of any of the human cases and no agglutination tests were performed. In the absence of these tests it cannot be accepted as established that this bacillus was the cause of the outbreak.

Other special bacilli have been occasionally described as the cause of outbreaks of illness from food.

Lubenau ([66]), for example, describes an outbreak in the Beelitz Sanatorium in July, 1906, affecting about 300 inmates. They had all eaten a special dish known as "Konigsberger Klops." There were no deaths. Bacteriological examination of this food showed *B. subtilis*. No examinations were made of the excreta of the cases, no agglutination tests were carried out, and on this slender evidence the outbreak was ascribed to *B. subtilis*. This is a quite unsubstantiated claim and the outbreak is only mentioned here as it is quoted by different writers as an outbreak due to this bacillus.

Parkes ([89]) describes an interesting series of cases of diarrhœa which continually occurred amongst the inmates of a large country mansion. The larder had recently been altered, and since then the meat and some other foods kept in the larder showed a pink growth. This was examined and identified with *B. prodigiosus*. The larder was cleaned out and disinfected and subsequently there was no diarrhœa or *B. prodigiosus* growth. As Dr. Parkes himself is careful to point out, the chain of evidence is incomplete and is only suggestive.

B. faecalis alkaligenes was shown by Ridder to be the probable cause of a single sporadic case of gastro-enteritis, but there is no evidence connecting it with an outbreak of food poisoning.

There is no clear evidence connecting etiologically *B. coli* or any of these other bacilli with outbreaks of food poisoning, although it is not possible definitely to exclude them as potential causes.

C. Ptomaine Poisoning and the Putrefactive Bacteria.

Until comparatively recently, amongst scientific medical men, and even now amongst the general public, the causation of meat poisoning has been ascribed to putrefactive changes in the food as eaten. This view, for example, was held by Flügge* who stated : "It appears that the wholesale poisoning of people so often observed, and which resembles severe epidemics of contagious diseases in its mode of appearance, is commonly caused by decomposing nutrient materials in the early stage of putrefaction. In cheese, sausages, meat or fish, to which these cases of poisoning are usually referred, the bases described above, or others yet unknown, have probably been formed under the action of bacteria from the albumen of the nutrient materials in question, and have occasioned their toxic symptoms."

Gradually, however, outbreak after outbreak, when carefully investigated, has been shown to be caused by meat, itself quite fresh and free from any putrefactive taint, but specifically infected by members of the Gaertner group of bacilli, (*see* Section V.). Putrefaction is still considered by many to play an important rôle in food poisoning although it is no longer advanced as the exclusive or even as the chief cause.

The bacteria concerned in putrefaction are numerous, but it cannot be said that any very complete and reliable combined bacteriological and chemical studies on the subject of putrefaction are available. Undoubtedly, some of the organisms concerned are aërobic and some anaërobic. Of the putrefactive organisms thought to be concerned in meat poisoning, suspicion has been most cast upon the proteus group, and to a lesser degree upon *B. coli* and its allies.

Chemical products of putrefaction.—The chemical products of putrefaction require brief consideration. Proteins being extremely complex bodies, the products which result from bacterial action upon them are very numerous, while many of them are themselves very complex.

The decomposition changes which occur proceed through a

* Micro-organisms, 1890, New Sydenham Society, p. 572.

number of stages, but these cannot be divided into a series of orderly sequences since they overlap, and several may be carried through simultaneously. The first stage is the solution and conversion of the proteins into proteoses and peptones. These are almost immediately broken down into various aromatic compounds (phenol, indol, skatol) with or without the intermediate production of tyrosine, leucine, and other amino-acids. Later, certain basic substances (amines) are formed which closely resemble the vegetable alkaloids. This group of substances has been called *ptomaines* by Selmi. In addition, a number of simple bodies such as ammonia, methane, carbon dioxide, hydrogen, &c., are formed as end products.

From the present point of view, the ptomaines are of the most interest. They have been extensively studied by Brieger and others. While some are quite non-toxic, others such as muscarin, sepsin, and tyrotoxicon are extremely poisonous.

The older conception of meat poisoning was that these ptomaines were the cause of the symptoms, hence the use of the term "ptomaine poisoning," which is still extensively current, particularly at coroners' inquests.

This view depends upon the assumption that food poisoning is due to the ingestion of decomposing food and upon the facts that from decomposing meat ptomaines have been isolated and that the injection of certain ptomaines causes in animals diarrhœa, vomiting and other symptoms commonly met with in food poisoning.

In the first place, it may be said that the methods by which ptomaines were isolated were inaccurate and pure bodies were not obtained. Later investigations have made it very probable that the poisonous symptoms caused by the injection of bodies obtained from nitrogenous putrefaction are not due to ptomaines. Bocklisch, for example, found that in preparing ptomaines from putrefying fish the most poisonous properties were possessed by the extraction fluid freshly prepared from putrefying broth. During the process of obtaining the bases the toxicity of the extract diminished.

Neither ptomaines nor the toxic bodies associated with them are produced in the early, but in the later stages of putrefaction when the meat would be far too nasty to eat. In the comparatively early stages of putrefaction at which food may be ingested it is most improbable that ptomaines or putrefactive toxins will have been produced in quantity sufficient to give rise to poisonous effects.

It must also be noted that the great majority of the animal experiments with ptomaines have been by inoculation and very few feeding experiments have been carried out. It is well known that even with toxins extremely pathogenic by inoculation, relatively enormous doses must be given by the mouth to produce any pathogenic effects.

The term "ptomaine poisoning" is clearly incorrect, but if a more modern nomenclature is adopted and we speak of the *toxins* produced by the putrefactive bacteria it still remains to be shown that they are a cause of meat poisoning.

Putrefactive Bacteria. — Professor Sidney Martin ([68], [69], [79]) has carried out a series of investigations upon the products of *Proteus vulgaris*. He found a toxic substance in the bodies of this bacillus readily extracted from the dried bodies by

distilled water. The physiological action of this substance in fatal or moderate doses was the production of a great lowering of body temperature in the rabbit, sometimes over 7° Fahr., the production of rapid evacuation of the intestinal contents and the causation of great bodily weakness, in some cases followed by collapse and death. This toxic body was found in the filtrate of broth cultures, diminishing in those over two weeks old, and is an excretion of the bacillus. It was not completely destroyed by heat, but the physiological effect was weakened.

Other products of this organism, which are more correctly described as putrefactive products, such as the aromatic alcoholic extract, and the foul smelling bodies which are removed by distillation, have but a slight physiological action.

Unfortunately Martin does not appear to have carried out any feeding experiments, and his investigations throw little light upon the power of *B. proteus* to produce toxins which can cause diarrhœa by feeding.

A number of earlier investigators have shown that *B. proteus* may be possessed of considerable pathogenicity when injected into small rodents. From the different experiments it would appear that this organism is one with very varying virulence, that any toxic symptoms are essentially in proportion to the dose and that pathogenic effects are due to toxins produced outside the body rather than to a true infection. Comparatively few feeding experiments appear to have been made with this organism, and the results are very conflicting. The following may be mentioned in addition to those referred to under the individual outbreaks recorded below.

Pfuhl ([96]) sterilized a piece of fresh beef and inoculated it from a broth culture of proteus. An abundant slimy over-growth was present on the meat after 48 hours' incubation. This meat and over-growth was used to feed a rat and a mouse. Both became very ill. The rat had quite recovered after five days the mouse died after 22 hours. The mouse *post-mortem* showed marked enteritis with liquid intestinal contents, enlarged liver and spleen. Proteus was isolated in pure culture from the liver and other organs. Obviously infection was very massive.

Metschnikoff ([76]), using *B. proteus* strains isolated from summer diarrhœa cases in young children, produced diarrhœa with fatal results in nursing rabbits fed with the bacilli. By feeding he also produced diarrhœa in young chimpanzees but no ill effects resulted when young macacus and cynocephalus monkeys were fed with these strains.

Herter and Broeck ([52]) carried out a few feeding experiments with two strains of *B. proteus vulgaris*. Young guinea pigs and nursing kittens were unaffected. Of three monkeys (*Macacus rhesus*) fed daily with 50 c.c. of broth cultures, two were unaffected even after a month's feeding, while the third showed diarrhœa with green stools. After 1½ months the animal became very weak and was killed. The walls of the large intestine were thickened, congested, and showed numerous small ulcerations. Cultures from the spleen and blood were sterile. The blood showed a slight agglutination reaction (1 to 50) to *B. proteus*.

Pergola ([91], [92]) isolated *B. proteus* from sausages which were associated with a small outbreak of illness at Lugo in Italy. The

bacillus isolated was very virulent to guineapigs, rabbits, mice and rats both by inoculation and by feeding. The outbreak is not described below as, apart from finding this virulent bacillus in the sausages, there is no evidence advanced that this organism had anything to do with the outbreak.

Jenson gives *B. proteus* as the cause of some forms of calf dysentery. In one epidemic he isolated *B. proteus* from the intestines but failed to isolate it from the blood or other internal organs. He found proteus organisms numerous in the intestinal contents, but they were not all the same strain. By feeding young calves with small quantities of broth cultures he set up symptoms of dysentery.

The last line of argument in favour of *B. proteus* and allied bacilli being a cause of food poisoning is the alleged existence of actual outbreaks due to these organisms. A small number of such outbreaks have been described and are usually quoted and re-copied from paper or book. In view of the practical importance of the matter it will be well to consider critically how far these recorded outbreaks may be really ascribed to ptomaines or putrefactive bacilli. The following is a brief summary of six of the most important and most frequently quoted outbreaks—

1. *Outbreak described by Levy* ([64]) *in* 1894.—Eighteen persons who frequented a certain restaurant were suddenly attacked with bloody diarrhœa, vomiting, depression and slight fever. Convalescence was very protracted. Several of the cases were very severe, and one patient died. The incubation period is not stated.

The outbreak was ascribed to *B. proteus* on the following grounds :—

a. The meat used was stored in a refrigerator. This had a strong odour of decomposition when examined and the floor was covered with a slimy brown crust which had an unpleasant stale odour and contained abundance of *B. proteus*. Both the raw and the cooked meat were kept for several days in the ice-chest.

b. In the fatal case proteus bacilli were isolated *post mortem* from the intestinal contents but *not* from the heart blood.

c. The proteus bacilli isolated were pathogenic to animals, and when *injected* caused hæmorrhagic diarrhœa with the presence of proteus in nearly pure culture in the stools.

d. The symptoms in the patients and in the inoculated animals were said to be similar. The *post-mortem* findings in the fatal case were similar to those shown in animals killed by sepsin.

It will be noted that no proof is advanced that *B. proteus* was present in or on the actual food eaten (this was, presumably, not examined); that the bacillus was not isolated from the fatal case apart from the intestine, the latter fact being of small significance, since this organism is a natural intestinal inhabitant, and that no animal-feeding experiments were carried out.

The outbreak is apparently ascribed to *B. proteus* because such

bacilli virulent on *injection* into laboratory animals were found in a situation where they could have contaminated the food which caused the outbreak. In view of the wide distribution of proteus this is wholly insufficient evidence.

2. *Mansfield. In August*, 1897. *Described by Wesenberg* ([144]).—63 persons were attacked after eating chopped-up meat derived from an emergency-slaughtered cow, the meat being eaten either raw or in the form of slightly-cooked liver. The cow suffered from a traumatic pericardial inflammation. The meat had been stored in a very damp cellar and piled up piece upon piece. It showed commencing decomposition.

A proteus bacillus was isolated from the meat, but as the latter already showed marked decomposition when it reached the laboratory this is not surprising. This bacillus when *injected* into mice and guinea-pigs was virulent.

Those who had eaten the food cooked remained unaffected. The symptoms were diarrhœa, headache, body pains, muscular weakness, vertigo and faintness. All recovered.

Here the diagnosis of proteus as the cause is based upon finding it in the meat in a condition which proved virulent on inoculation.

3. *Outbreak described by Glücksmann* ([46]) *in March*, 1898.—Two persons (father and son) fell ill a few hours after eating a little half-smoked pork derived from a pig slaughtered on account of disease (nature not ascertained). One of the patients died.

The smoked meat was only examined four days after the fatal meal, and it had then undergone decomposition. When examined, colonies of *B. proteus vulgaris* were very abundant. Broth cultures of the isolated proteus *injected* into mice and guinea-pigs killed them with symptoms of diarrhœa.

Here, of course, there is no evidence at all of a proteus infection.

4. *Outbreak in Switzerland described by Silberschmidt.*—In September, 1898. Forty-five cases due to the consumption of one kind of sausages (so-called "Landjäger"). The symptoms, for the most part, came on about twenty-four hours after eating. The chief symptoms were very profuse diarrhœa, severe cramp-like body pains, fever—often marked—vomiting in most cases and marked prostration. The duration of the disease was considerable, most of the cases being about two weeks under treatment. One death occurred two and a half days after eating. No bacteriological investigation of the patients was made. Apart from a rancid taste, the sausage meat appeared normal. *B. proteus vulgaris* and a coli-like bacillus were isolated from the sausage meat.

Some feeding experiments were made, but they were quite inconclusive. Mice and guinea-pigs fed with dry pieces of sausage were unaffected, but when pieces of the sausage were kept for several days in broth and then fed to mice and guinea-pigs, the latter died and *B. proteus* was isolated from the intestinal content, but not from the organs.

Here, obviously, there is no evidence that proteus was the cause of the outbreak.

5. *Hanover. 1900.*—Described by Pfuhl ([96]). During the night of April 12th, 1900, 81 soldiers were suddenly attacked with acute

gastro-enteritis, 4 to 12 hours after eating sausage meat. The symptoms rapidly subsided.

The sausage meat was not packed into skins, but into jars. It was prepared on April 11th from heart, lungs and stomach of three different animals. It was cooled in the preparation-room. The sausage-meat, in appearance, taste and smell, was perfectly normal. No evidence of putrefactive products could be obtained by injecting animals with alcoholic and ether extracts. No information was available as to the health of the animals from which the meat was derived.

The bacteriological examination of the sausage meat showed, in addition to other bacteria, *B. proteus* in abundance.

Two rats and two mice were fed with the sausage. The rats each received about ten grammes and were ill, suffering from diarrhœa, but recovered. The mice received about five grammes and were also ill with diarrhœa. One was killed twenty-four hours after feeding and *B. proteus* (*P. mirabile*, *Hauser*) was isolated from the blood and internal organs, cocci and a sarcina being also present. The other mouse died three days after feeding, and a proteus strain was isolated from the internal organs. The proteus isolated from this mouse was slightly virulent.

While there is slightly more evidence in this outbreak pointing to proteus toxins as the cause, there is no etiological connection established as the human cases do not appear to have been bacteriologically investigated.

The feeding experiments do not prove much since it is not uncommon to recover *B. proteus*, *B. coli*, &c., from mice which have died after feeding with foods such as sausage meat. Schellhorn ([115]) for example fed a large number of mice with germ-free meat and 50 per cent. died, usually within 3 to 5 days. 115 mice died in this way. As a rule no bacilli were found *post mortem* but in a few cases *B. coli* were found. *B. proteus* was not isolated by him from any of the mice.

The sausage meat in Pfuhl's cases was prepared from stomach, &c., so the presence of proteus in it is of no special significance, and even if the strain present was toxic to mice when administered in sausage meat, this fact would not necessarily indicate an etiological connection between the sausage meat and the outbreak.

On other grounds, it is unlikely that *B. proteus* was the cause of illness in these cases. The rapid onset and quick subsidence point either to chemical poisons or to toxins only. It is improbable that the poisonous elements in this meat were due to *B. proteus*, since the meat was quite healthy in appearance, odour, &c., and there does not appear to have been sufficient time for any products of the kind to have been formed.

It is more likely that the outbreak was due to heat-resistant toxins, the product of some other organism, the organism itself having been killed in the process of preparing the sausages. The feeding experiments do not negative this, since the heat-resisting toxins might kill the mice and allow secondary invasion by intestinal bacilli, such as proteus.

The report does not say when the sausage meat was examined. If not examined immediately the presence of proteus is easily accounted for.

6. *Hanover. 1901* — Described by Schumburg ([121]). In May, 1901, thirty-four persons became ill after eating sausage. The symptoms came on after a few hours and consisted of nausea, profuse diarrhœa, repeated vomiting, weakness and debility. In most of the cases the symptoms subsided after twelve hours, and only in one or two persons did they continue until next day.

The sausage meat was not put into skins but was used loose. When bacteriologically examined it showed two kinds of bacteria—one a member of the potato bacillus group (a harmless saprophyte), the other proteus. A rat and two mice fed with the sausage meat died after 24 hours and proteus was isolated from the spleen, liver and heart blood, and was abundently present in the intestinal contents. The strain isolated from these animals was virulent when fed to mice and rats.

Apparently no examinations at all were made of the patients, and the proof rests solely upon finding a *B. proteus* in the food which was virulent to mice and rats.

From the summary of the outbreaks given above it is evident that for none of them was it established that *B. proteus* was etiologically concerned, and it may be said that available evidence is insufficient to establish that this organism is ever a cause of extensive outbreaks of food poisoning.

Dealing with putrefaction, using the term comprehensively and without attempting to restrict its effects to the products of *B. proteus* or any single bacillus, it still remains a matter of controversy as to how far its occurrence in meat may be a cause of human disease.

As a cause of extensive widespread outbreaks putrefaction can certainly be excluded, but apart from these there still remains a reasonable probability that individual cases and limited outbreaks may be due to the consumption of meat in a state of early decomposition. As already pointed out, the changes which occur in the initial stages of the putrefaction of meat are very complex, and while the conception that the symptoms are due to ptomaines must be abandoned, it is probable that other toxic substances are produced which on ingestion with the food are liable to cause gastro-intestinal disturbances which may be of considerable severity.

It is very desirable that investigations upon this aspect of the subject should be carried out on modern bacteriological and chemical lines.

D. Botulism.

This interesting but fortunately rare type of food poisoning has been recognised for nearly a century, but knowledge as to its true pathology only dates from 1895. Kerner in 1820 reported upon a number of epidemics in various parts of Würtemberg and in 1822 reported a further 98 cases with 34 deaths. Since these dates many other outbreaks have been reported, the greater number occurring in Würtemberg or its neighbourhood. The majority have been due to eating sausage and the condition is often spoken of as "sausage poisoning."

Outbreaks of botulism are now of very rare occurrence anywhere and I have been unable to trace the occurrence of a single outbreak in Great Britain. Only a brief description of the condition is therefore required.

The bacterial origin of this variety of food poisoning was ascertained by Van Ermengem, who in 1895 isolated *B. botulinus* from a ham, the consumption of which had caused 50 cases of botulism at Ellezelles (Belgium) in December, 1895. Three of the patients died, and Van Ermengem also isolated the bacillus from the spleen and gastric contents of one of the fatal cases.

B. botulinus is a large spore-bearing bacillus which will only grow when oxygen is strictly excluded (obligatory anaërobe). Its most characteristic property is the production of extremely powerful toxins which reproduce, when injected into, or fed to, suitable animals, the characteristic symptoms of the disease. The bacillus, like the tetanus bacillus, will not grow to any extent in the animal body, but produces its poisonous effects by the toxins which it excretes into the nutrient material in which it is growing.

The toxin is not heat resistant, being destroyed by exposure for half-an-hour at 80° C., while it is quickly destroyed by light and air. The spores are also not highly resistant.

The bacilli, according to Römer, grow best at 22° C., while they will not grow upon media, having an acid reaction. Cultures have a rancid butyric acid odour.

The symptoms exhibited in cases of botulism are very characteristic and strikingly different from those met with in ordinary cases of food poisoning. They are almost entirely referable to lesions of the central nervous system. Prominent symptoms, according to Van Ermengem, are paralysis of accommodation, mydriasis, ptosis, double vision, dryness of the mouth and throat, aphonia, dysphagia, retention of urine. Fever is absent and consciousness is not impaired. Vomiting, diarrhœa and other disturbances of the gastro-intestinal tract are frequently absent or, if present, are slight and transitory. The symptoms come on 24 to 36 hours after eating the meal. Death, due to bulbar-paralysis, occurs in 25 to 30 per cent. of the cases. Outbreaks have most frequently followed the consumption of liver and blood sausages but have also occurred after eating pickled or smoked meat, tinned meat, preserved foods, &c.

In none of the outbreaks have any of these foods been eaten in the fresh state, the toxic properties only developing after the food has been stored. It is quite clear that the development of toxic properties is due to an invasion of the food by *B. botulinus* from outside sources, and this can only occur when suitable anaërobic conditions are provided. In the Ellezelles outbreak the ham from which Van Ermengem isolated the bacillus had been laid at the bottom of the pickling cask and covered with the pickling solution, the other ham from the same animal, which was quite harmless, being laid above it and not covered by the liquid. The poisonous ham was not putrid but had a strong rancid odour.

In an outbreak described by Römer, which was traced to the consumption of ham, the ham had also been placed in brine.

The infected foods have usually been eaten in a raw condition, and Van Ermengem states that this disease has never been caused when the food has been properly cooked.

Information is very scanty as to the source of *B. botulinus*. Van Ermengem was unable to find it in the intestinal contents of pigs, cows, &c., or in manure or soil. Fifty-two different samples were examined.

The only positive finding with which I am acquainted is that of Kempner and Pollack, who isolated a bacillus from the excreta of a healthy pig which agreed in all its characters, including toxicity, with *B. botulinus*.

The prevention of the conditions favourable to the infection and growth of *B. botulinus* should not be difficult. Sausages and other forms of prepared food must be manufactured only from good and clean materials, while all such foods should be properly cooked, either in course of manufacture or before being eaten. Van Ermengem also recommends that in salting, the brine should contain at least 15 per cent. sodium chloride since *B. botulinus* will not develop in media containing more than 6 per cent. sodium chloride.

SECTION V.

The Gaertner Group of Bacilli in relation to Food Poisoning.

As shown in Section IV., the majority of recorded outbreaks are caused by bacteria of the Gaertner group. A proper understanding of the etiology of food poisoning is impossible unless careful attention is paid to the distribution in nature, pathological properties and physical characteristics of these bacilli. These important matters are considered in the following sub-sections :—

A.—Gaertner group bacilli as disease-producing organisms.
B.—Classification of Gaertner group bacilli.
C.—Distribution of Gaertner bacilli in nature.
D.—Viability, vitality and other physical properties of the Gaertner bacilli and their toxins.

A. *Gaertner group bacilli as disease-producing organisms.*

It is very convenient to group together *B. enteritidis*, *B. paratyphosus B* and other organisms with identical cultural characters as the Gaertner group of bacteria and to include with them organisms closely allied but which can be distinguished by an extended series of cultural tests (such as the power to ferment salicin, glycerin, &c.). The latter group of organisms may be conveniently spoken of as pseudo-Gaertner or perhaps better as para-Gaertner bacilli. As far as our knowledge goes, the para-Gaertner bacilli are not a cause of either human or animal disease, but the true Gaertner bacilli are etiogically associated with a number of diseases. These must be briefly considered.

In man, Gaertner group organisms are associated with two conditions, viz., outbreaks of food poisoning and cases of paratyphoid fever.

Paratyphoid fever is a comparatively rare disease in this country, although it is undoubtedly more prevalent than the recorded cases might suggest. Unless bacteriological tests are used it is not possible to differentiate it from typhoid fever. The symptoms and

clinical features are nearly indistinguishable, although the symptoms are usually less severe and the fatality rate is considerably lower in paratyphoid fever. Like typhoid fever there is no evidence that paratyphoid fever occurs in the lower animals.

A very few cases in this country, but more in India and perhaps others abroad, are due to *B. paratyphosus A*, a closely allied organism which can, however, be culturally distinguished from the the true Gaertner group organisms by its failure to produce alkalinity in milk.

In almost all the cases of paratyphoid fever in this country the associated bacillus is *B. paratyphosus B*, a true Gaertner group organism, but one which, as will be shown later, is not concerned with the causation of food poisoning.

In animals, members of the Gaertner group appear to play a considerable disease-producing rôle, and in this way are sharply separated from the allied typhoid bacillus which is not, as far as is known, a natural disease-producing organism in animals. The diseases and conditions in animals associated with one or other member of the Gaertner group require detailed consideration.

1. *Swine Fever.*—A member of the Gaertner group, *B. suipestifer* was accepted for many years as the cause of this disease, but in 1903 and 1904 the work of De Schweinetz and Dorset and of Dorset, Bolton and McBryde ([36]) showed clearly that this disease was due to a filterable virus and that *B. suipestifer* plays only a secondary part. Their work was confirmed by Ostertag and again later by Uhlenhuth, Hübener, Xylander and Bohtz in 1908 ([133]).

It would appear, however, that *B. suipestifer* is not a mere passive concomitant but has a disease producing rôle.

In 80 cases of swine fever Preiss found this organism 31 times (38·8 per cent.), and Uhlenhuth, Hübener, Xylander and Bohtz 76 times from 171 infected animals (45·4 per cent.).

Uhlenhuth and his co-workers ([136]) found that *B. suipestifer* is not the only organism which secondarily invades the organs of swine fever infected animals. They found that different strains of bacilli predominated during different periods. Thus for a long time the organs of the animals dying in their experimental disease sty contained chiefly *B. suipestifer*. Then followed a time during which this strain was but seldom found, and almost all the animals that died showed *B. enteritidis* while later on strains which resembled *B. paratyphosus B*, but which were not agglutinated by paratyphoid serum, predominated.

2. *Septicaemia and other diseases of calves.*—It would appear that calf septicaemia, dysentery and pneumonia are caused by a number of different kinds of bacteria, but that in a good many cases Gaertner group bacilli are either the cause or are found to be present.

Thomassen ([127]) in 1897 described a fatal septicaemia in calves in the neighbourhood of Utrecht and isolated *B. septicus vitulorum* from the spleen, kidneys and other organs of the affected animals. This organism has been subsequently identified with *B. enteritidis*.

Uhlenhuth and Hübener have shown that Jensen's paracolon bacillus described by him as the cause of calf dysentery is, in at least many cases, a true Gaertner group bacillus.

Zeller ([149]) between 1902 and 1907 isolated 40 Gaertner strains from cadavers of diseased calves which died or were emergency slaughtered. The calves suffered from septicæmia, enteritis, lung inflammation, &c.

Titze and Weichel ([129]) found Gaertner group bacilli in cases of white scour in calves.

Schmidt ([120]) examined the cadavers of 61 dead or emergency slaughtered calves and the intestinal contents of 12 others. The calves suffered from septicæmia, dysentery, pneumonia, &c. Gaertner group bacilli were isolated from nine cases. He also showed in further investigations that the bacilli were present during life in the blood of the calves.

Riemer ([101]) in 4 out of 11 cases of septic disease in calves isolated true Gaertner group bacilli.

Winzer ([147]) examined the carcases of 30 calves showing evidence of septicæmia. In eight of these cases Gaertner group bacteria were found. The animals were all quite young. The bacilli were chiefly present in the spleen, less commonly in the glands or liver, and only occasionally in the musculature. The isolated organisms were pathogenic to mice. Winzer, in addition, made numerous examinations of the carcases of full-grown animals, but in no case could he find Gaertner group bacilli.

3. *Pyaemic and septicaemic conditions in the domestic animals generally.*—Bollinger in 1876 first drew attention to the frequency of the association of food poisoning outbreaks in man with the consumption of the meat of animals suffering from such diseases. Basenau in 1893 isolated *B. morbificans bovis* from the muscles and organs of a cow emergency slaughtered on account of puerperal metritis. He subsequently on two occasions isolated bacilli closely allied to this organism from animals ill from septic disease.

Zwick ([153]) investigated 21 cases of acute mastitis in cows and isolated Gaertner bacilli from two of them. In the second case *B. enteritidis* was isolated from the udder secretion in pure culture. The animal gradually became better, and, after the birth of the next calf, lactation came on again in the diseased quarter. Zwick unfortunately does not say how long *B. enteritidis* persisted, or whether he made any investigations in this direction.

Fischer in 1896 isolated *B. enteritidis* from the spleen of a cow which had died or been slaughtered on account of udder inflammation.

On the other hand, in opposition to this conception, we have the fact that a number of investigators have examined the meat of a large series of animals suspected to be suffering from septicaemic diseases, and have found Gaertner group bacilli only in a very few instances. Müller ([83]), for example, examined an extended series of specimens from slaughtered cattle, examining both the muscular tissue and the internal organs. While the majority were from healthy animals, 124 specimens were from cattle which had been subjected to emergency slaughter on account of septicæmia, while a number were from animals which, after slaughter, showed signs of septicæmia on examination. In only one case out of 110 animals did he find a Gaertner group bacillus, and it is of interest to note that the consumption of the meat of this animal caused a meat poisoning outbreak involving 97 persons. Not only was the flesh normal in

appearance, but the organs showed no evidence of disease to the naked eye.

Necrotic foci are mentioned fairly frequently as being present in the liver, spleen and kidneys of animals, particularly calves, slaughtered in continental abattoirs. Gaertner bacilli have been sometimes found in these necrotic areas.

4. *Enteritis in Cows.*—In a good many meat-poisoning outbreaks the meat has been obtained from a cow suffering from enteritis. Cases of enteritis in cows, apart from those associated with food-poisoning outbreaks, in which Gaertner group bacilli have been isolated appear to be rare. Mohler and Buckley ([77]) describe one such outbreak. Seven out of 21 cows in a cowshed suffered from enteritis and died, while three others exhibited early symptoms but recovered. A Gaertner bacillus was isolated from all the fatal cases. It is interesting to note that one cow apparently recovered from the acute attack, but ultimately died 26 days after the onset.

5. *Certain Diseases of Birds.*—There have been a number of epidemics of disease (*Psittacosis*) in parrots due to a true Gaertner group bacillus (*B. psittacosis*) isolated by Nocard in 1893. Nocard isolated the bacillus from the bone marrow of birds which had died on the journey from Buenos Ayres. In subsequent epidemics this bacillus has been isolated from both the diseased parrots and from the blood of the human cases.

From the parrots the disease has spread to man, and in April, 1892, an extended outbreak occurred in Paris of 42 known cases with 14 deaths.

Tartakowsky has described an infectious enteritis in sparrows due to a Gaertner bacillus.

Joest isolated a bacillus, apparently a Gaertner group organism, from a canary suffering, with others, from an epidemic disease associated with catarrhal enteritis and splenic tumour.

6. *Diseases amongst Rodents.*—*B. typhi murium* was isolated by Löffler as the cause of an epidemic in mice, and has subsequently been isolated from other mice epidemics. It has been used as living poison to set up an epizootic among mice and so cause their extermination.

In the same way a number of Gaertner group bacilli have been used to set up infective disease in rats. Of these may be mentioned Danysz's bacillus, Dunbar's bacillus, and "Ratin."

The first-named bacillus was isolated by Danysz ([31]) from a naturally caused epidemic amongst harvest mice. He increased its pathogenicity by special animal passage methods so that it became pathogenic for rats. It has since then been extensively employed to exterminate rats and mice. For example, Danysz ([32]) states that in 1903–04 nearly 600,000 litres of cultures of this bacillus were distributed in France for the purpose of destroying field mice. From the description in the paper quoted, it is evident the cultures must be of low virulence to man, since during the distribution of the virus on a large scale abundant opportunities for human infection were afforded.

Dunbar's bacillus was isolated from a rat epizootic in Hamburg. "Ratin" was a bacillus isolated by Neuman from the urine of a two-year-old child suffering from cystitis.

Spontaneous outbreaks of infectious disease amongst rats and mice appear to be not uncommon. They certainly are not infrequent in laboratory animals, and three definite outbreaks have occurred in my laboratory mice, two due to, or associated with, *B. enteritidis* and the third to another Gaertner organism, the precise sub-group not being investigated.

Outbreaks of disease due to Gaertner infection have been met with in guinea-pigs, but in one outbreak recorded by Petrie and O'Brien ([93]) they were only present as associated organisms, the cause of the outbreak being some unrecognised filterable virus.

Outbreaks of disease in other animals may possibly have been due to Gaertner group bacilli, but the bacilli have not been sufficiently worked out. Mori ([81]), for example, describes a bacillus isolated from a cat which, with several others, had died from an epizootic in Siena. The characters given are insufficient to indicate whether it was a Gaertner organism or not.

From the above summary it is clear that the Gaertner group is an important disease-producing factor in animals as well as in man. While there is no doubt as to the association of the Gaertner bacilli with the diseases and conditions described, it may be that they do not always play the primary rôle, and possibly in other diseases as well as swine fever, they may only be present as a secondary infection.

B. *Classification of Gaertner group bacilli.*

Considerable confusion in the nomenclature of this group has existed owing on the one hand to each bacillus isolated from outbreak or animal disease being frequently given a special name, and on the other to insufficient care being taken fully to describe and work out isolated bacilli of the Gaertner group.

The Gaertner group itself is really a sub-group of the large coli-typhoid group, the members standing in their cultural characters between the chemically active coli group and the chemically rather inactive typhoid group. The group itself may be conveniently divided into two sub-groups—

Sub-group A.—True Gaertner bacilli.

„ „ B.—Para-Gaertner bacilli (or pseudo-Gaertner bacilli).

The bacilli of the para-Gaertner sub-group are a number of organisms, for the most part unnamed, which appear to be not very uncommon in the healthy animal and human intestine, and which are of chief interest from their close resemblance to true Gaertner bacilli. I first drew attention to their presence in my reports on this group to the Local Government Board ([109, 110, 112]).

They can only be culturally differentiated from the true Gaertner organisms by an extended series of fermentation tests, while they fail to be agglutinated by the sera obtained by immunizing animals with any of the members of the true Gaertner sub-group. They are also for the most part non-pathogenic.

They have not so far been found as a cause of disease in man or in animals.

The true Gaertner bacilli are culturally indistinguishable but can be differentiated by means of agglutination and other serological tests into several organisms.

The following classification may be adopted :—

1. *B. enteritidis.*—Includes not only many of the Gaertner bacilli isolated from food-poisoning outbreaks, but also most of the rat viruses, some of the strains of *B. typhi murium* and Thomassen's *B. septicus vitulorum.*

2. *B. suipestifer.*—Includes *B. suipestifer* itself, some of the *B. typhi murium* strains, probably *B. psittacosis*, and all the food poisoning bacilli of this group which are not *B. enteritidis.*

3. *B. paratyphosus B.*—The bacillus associated with most cases of paratyphoid fever and also found in the human intestine in carrier cases associated with this disease.

4. *B. morbificans bovis.*—This organism, (see page 31) isolated by Basenau, may be identical with either (1) or (2) but it is difficult to place it accurately.

As regards *B. paratyphosus B* there is a decided difference of opinion as to whether this organism is identical with *B. suipestifer.* With but very few exceptions the German view is that these two strains are identical. English investigators have for the most part found differences. Of the English workers particular attention may be directed to the work of Bainbridge and O'Brien ([9]) who have published an extended series of absorption tests which show clearly that these two organisms can be differentiated.

C. *Distribution of Gaertner group bacilli in nature, apart from pathological conditions.*

A good deal of work has been carried out within the last few years upon the natural distribution of Gaertner group bacilli, the results obtained being far from concordant. The available data as to distribution may be conveniently grouped as follows—

a. In the human intestine.
b. In the animal intestine.
c. In meat, particularly prepared meats.

a. In the human intestine.—B. paratyphosus B is found in the intestine in cases of paratyphoid fever and in persons who have comparatively recently had an attack of paratyphoid fever and are acting as acute or chronic carriers.

Apart from these cases all the English investigators have failed to find that *B. paratyphosus B* or other true Gaertner group organisms are natural inhabitants of the healthy human intestine.

In 1908–09 I ([112]) examined very carefully and by methods of proved utility 53 specimens of human excreta from healthy persons or from persons suffering from disease, a few being cases of paratyphoid fever. Apart from finding *B. paratyphosus B* in actual cases of that disease only one organism of the true Gaertner group was isolated, a *B. enteritidis.*

Morgan ([79]) examined about 300 cases of summer diarrhœa and only found two true Gaertner organisms, both *B. suipestifer.*

Williams, Murray and Rundle ([146]) examined the stools of 100 normal children and failed to find any true Gaertner group organisms.

Bainbridge and O'Brien ([9]) examined the fæces and urine of

numerous typhoid convalescents and failed to isolate any true Gaertner group bacilli.

Lewis ([65]) examined 214 specimens of fæces from 146 normal children and 70 specimens of fæces from 61 cases of diarrhœa in infants. Only *one* strain identical with true Gaertner group bacilli was isolated. It was pathogenic but the precise sub-group was not determined.

The German results are conflicting. Conradi, Prigge and Sachsmüke and others recorded *B. paratyphosus B* as present in the excreta of a considerable proportion of typhoid convalescents and healthy persons, while Sieffert, Sobernheim, Aumann and others do not confirm these observations.

Aumann's ([2]) figures include 799 specimens of human excreta, urine, blood or vomit examined in the Municipal Hamburg Hygienic Institute 1905–1910. Of these 498 were excreta specimens and 6 per cent. (31 specimens) showed true Gaertner group bacilli, 13 being paratyphoid bacilli and 18 *B. enteritidis*. In all but one the presence of these bacilli was closely associated with epidemics or cases of disease. In the 301 other specimens 3 paratyphoid B bacilli were isolated, in each instance from a case of paratyphoid fever.

b. In the animal intestine.—In view of the fact that fæcal contamination of food during and sometimes after preparation is common, it becomes of cardinal importance to ascertain to what extent bacilli of the Gaertner group occur in the intestines of healthy animals. The domestic animals used for food are naturally the most important in this connection.

Uhlenhuth Hübener, Xylander and Bohtz ([133]) investigated bacteriologically the intestinal contents of 600 pigs from the Central Berlin slaughter house. Although they only examined one loopful of the intestinal contents, they isolated *B. suipestifer* from 8·4 per cent. (51 cases) of the animals.

In confirmation of these results Seiffert found the bacilli twice in 60 pigs, Eckert in 4 out of 10 pigs and Grabert in 7 out of 23 pigs.

The cultural tests which these investigators employed and which were accepted by them as sufficient are not adequate to distinguish the bacilli from the para-Gaertner bacilli which I have shown to be not infrequently present in the healthy animal gut. These investigators for example did not test the fermentation properties of their organisms upon dulcite, salicin or saccharose.

Some virulence tests—subcutaneous inoculation of mice—were performed and the mice succumbed. The strains were agglutinated by hog-cholera sera but the exact dilutions employed are not given.

In healthy calves and sheep Uhlenhuth, Hübener and Andreijew found organisms in small numbers which were possibly paratyphoid bacilli. Andreijew ([3]) is also frequently credited with finding true Gaertner bacilli in sheep intestines, but what he did find was bacilli "which in their cultural and biological characters could be, with more or less probability, added to the hog-cholera group of bacilli." Para-Gaertner bacilli are not uncommon in sheep.

In view of the insufficient cultural tests used, these positive German findings cannot be unreservedly accepted since they do not

differentiate the bacilli isolated from the para-Gaertner bacilli which are scanty but natural intestinal inhabitants and which have nothing to do with food poisoning.

Also, on the other hand, there are a number of negative German findings. For example, Titze and Weichel ([129]) examined the excreta of 44 cattle, 60 calves, 15 sheep, 3 goats, 16 dogs, 24 rabbits, 50 guinea-pigs, 13 fowls, 14 pigeons, 6 geese and 14 sparrows with quite negative results except that in the excreta of one horse they found a paratyphoid bacillus.

Eckert also examined the intestinal contents of 12 horses, 16 cattle, 8 sheep, 1 goat and 5 rats, and only in the case of one horse did he find a true Gaertner group bacillus.

Aumann examined the excreta of 200 recently slaughtered animals, consisting of 101 pigs, 48 cattle, 41 sheep and 10 calves, and failed to find true Gaertner group bacilli in any of them.

Only a few English investigations appear to have been carried out, but their results are entirely opposed to the view that these organisms are natural intestinal inhabitants.

Morgan ([78]) examined the fæces and intestinal contents of a number of animals and isolated 21 cultures of bacilli of the Gaertner type. They were, however, not isolated by direct cultivation from the intestinal contents but only from animals inoculated with the material under examination, a method which is now admitted to be unreliable for this purpose.

In 1906–1908 I ([109, 110]) examined with great care (for example over 700 plates were inoculated) the intestinal contents of 26 animals and 5 specimens of animal excreta, the animals selected being 7 pigs, 6 bullocks or cows, 5 calves, 4 sheep, 8 horses and 1 mouse. No true Gaertner group organisms could be found, although a considerable number of para-Gaertner bacilli were isolated which culturally resembled true Gaertner bacilli extremely closely.

These results have been confirmed by Bainbridge ([7]) who examined the intestinal contents of 50 pigs without finding any true Gaertner group bacilli.

Apart from animals used for animal food, evidence has been adduced that true Gaertner bacilli may be met with in the intestinal contents of rats and mice.

Heuser ([53]) examined 100 mice and in 5 isolated Gaertner bacilli of both types, *i.e.*, *B. enteritidis* and *B. paratyphosus B* (*vel B. suipestifer* according to German classification) from their intestinal contents. The mice showed no symptoms of disease. He also examined about 60 white rats and found *B. enteritidis* but no *B. suipestifer*. He found that by animal passage bacilli of the *B. suipestifer* group showed a rise of virulence to rats.

Zwick and Weichel ([155]) examined 177 mice and in 28 found Gaertner group bacilli present.

There is further evidence as to the occurrence of true Gaertner group bacilli in the intestines of rats and mice from certain feeding experiments. Mühlens, Dahm and Fürst ([82]) fed mice with different kinds of prepared food and a number of them died, Gaertner group bacilli being isolated. Gaertner group bacilli were not found in the foods by cultural tests. Zwick and Weichel fed 140 white mice with 70 samples of salt meat and different forms of

pig, all of which culturally examined failed to show Gaertner group bacilli. 85 (60·7 per cent.) of the mice died, and from two of them *B. paratyphosus B* was isolated.

These feeding experiments suggest that the Gaertner bacilli were present in the mice, probably in the intestines, and under the conditions of the experiments invaded the body and caused a general nfection with death.

It has already been recorded that rats and mice suffer from epidemics of disease due to true Gaertner group bacilli.

While these facts are advanced by certain observers as showing that Gaertner group bacilli are natural intestinal inhabitants of rats and mice a more probable explanation is that they are present in these cases as carriers and are not really natural inhabitants.

In outbreaks amongst rats and mice due to Gaertner bacilli, while many of the animals die, a considerable percentage recover or even do not suffer at all. There is a strong probability that these will act as carrier cases for some time.*

Rats fed with rat virus often recover, and are then highly immune and can eat large amounts of virulent virus without any apparent ill effects. The ingested bacilli are present in large numbers in the excreta.

Rats frequently infest slaughter-houses, so that the bacterial contents of the intestines of rats and mice is of practical moment.

Petrie and O'Brien ([94]) succeeded in experimentally producing the carrier-state in guinea-pigs by feeding them with *B. suipestifer*. In the case of two animals (out of six fed) the bacilli were isolated from the faeces for as long as 59 days after the last feeding. The authors do not state if the bacillus used was virulent by injection when used for feeding, or if any impairment of virulence resulted from the sojourn in the alimentary tract of the guinea-pigs.

In an epizootic amongst the stock guinea-pigs at the Lister Institute due to *B. suipestifer*, recorded by O'Brien ([88]), the survivors showed definite immunity to this bacillus, and five of them proved to be carriers, excreting the bacillus intermittently five months later.

Bainbridge ([8]), in the Milroy Lectures for 1912, also draws attention to the importance of the rat in relation to meat-poisoning, and suggests that the rat's intestine may be the true home of *B. enteritidis*, and that it reaches the alimentary canal of cattle and other domestic animals through the contamination of their food or bedding by rats.

c. In meat, particularly prepared meats.—The only investigations which appear to have been carried out in this country are my own ([113]) for the Local Government Board in 1909. Sixty-four specimens of brawn, sausage, tinned and other prepared foods were examined,

* An investigation upon the presence of Gaertner group bacilli in rats, upon which I am at present engaged with the help of Mr. W. J. Read, supplies further evidence in support of this supposition. Up to the present twelve apparently healthy rats have been examined by us, and while we have found true Gaertner bacilli (*B. enteritidis*) in three of them, in each case the bacilli were isolated from the spleen or liver and could not be isolated from the intestinal contents. In other words, they were probably present as the result of an old infection from which the rats had recovered.

also 18 samples of pickling fluids from various sources. While pseudo- or para-Gaertner organisms were not uncommon, from only one sausage (out of 28) was a true Gaertner organism isolated. This was *B. enteritidis* and not *B. suipestifer*, although the sausage was a pork one. The particular sausage from which it was isolated was obtained from a small local butcher who made his sausages on the premises. No illness was known to result from consumption of the rest of the sausages.

On the whole the German investigations, which have been fairly numerous since 1908, are in accord with and confirm these results, but several workers have found the bacilli more frequently.

Mühlens, Dahm and Fürst ([82]) examined 57 samples of different kinds of prepared meats bought in Berlin shops, and including pickled goose-breast, raw ham, cooked ham, smoked ham, smoked tongues, &c. In no case could they find any Gaertner group bacilli by cultural examination. When they fed white mice with the different foods, a large number of the mice died, and Gaertner group bacilli were isolated. Since, however, as already pointed out, mice feeding and inoculation methods are useless for this purpose, the essential value of their work is that they failed to find Gaertner group bacilli in these prepared foods by cultural examination.

Hübener examined 100 different varieties of sausage and isolated six Gaertner group bacilli, two from fresh blood sausages and four from smoked sausages. The cultural characters of the bacilli are not given.

Rimpau records that he found *B. paratyphosus B* in a liver sausage quite free from objectionable appearance or odour, and the use of which was in no way harmful.

Rommeler examined 51 samples of sausage, and in eight found paratyphoid bacilli, while from eight samples of "hackfleisch" in five he isolated the same bacillus. As no cultural, agglutination or inoculative experiments are given, it is not possible to say if he differentiated the true from the para-Gaertner forms.

Trautmann examined 51 samples of sausage, ham, goose-breast and smoked meat without finding any Gaertner group bacilli.

Zwick and Weichel culturally examined 70 samples of salt meat, goose-breast and different forms of prepared pig meat, and were unable to find any Gaertner bacilli.

Aumann investigated 150 specimens of sausage from Hamburg shops and failed to find Gaertner group bacilli in any of them.

Zweifel at Leipzig examined 248 specimens of chopped meat (hackfleisch), obtained quite fresh, both by direct plating and by enrichment method. In no case did he find true Gaertner group bacilli.

These results may be taken as showing that true Gaertner group bacilli are nearly always absent from prepared meat foods, but that very occasionally they are present.

D. *Virulence, Viability, Vitality, &c., of Gaertner Group Bacilli.*

True Gaertner bacilli are in general possessed of considerable pathogenicity for the ordinary laboratory animals. In this respect

there is no essential difference between the different members, *i.e.*, *B. enteritidis*, *B. suipestifer*, *B. paratyphosus B.*

The toxins to which their pathogenic properties are due are almost entirely endo-toxins. Cathcart ([28]), for example, found, when the bacilli were grown in broth and the bacilli filtered off by means of a Chamberland filter, that cultures up to three days old yielded practically no toxin, and although filtrates of older cultures showed varying degrees of toxicity, these were probably due to disintegration of the bacilli after death. The most toxic preparations were obtained by autolysis of the bacilli. Cathcart also showed that *B. paratyphosus B* cultures yielded an endo-toxin quite as toxic as that obtained from *B. enteritidis*.

Of the laboratory animals, mice are particularly sensitive to inoculation with Gaertner group bacilli, while, particularly when recently isolated cultures are used, guinea-pigs, rabbits and rats usually die one to two days after inoculation, subcutaneous or intraperitoneal.

Two facts are, however, of considerable significance. One is the varying virulence of these bacilli and the other is the greatly diminished toxicity when the method of introducing the bacilli is by feeding.

In old laboratory cultures, virulence is often greatly diminished and may even be altogether absent. Gaertner noted that while the infective power of the bacillus may not be greatly impaired, the bacillus rather rapidly loses its power to produce toxins. Later observers have confirmed this. Holst found, using the bacillus isolated by him from the Gaustad Asylum outbreak, that the toxin-producing capacity, after rapidly diminishing, could be restored by several passages of the bacillus through pigeons. Van Ermengem found that the bacillus isolated by him from the Moorseele outbreak (*B. enteritidis*) behaved in the same way.

We have but little evidence as to any alteration of virulence in carrier cases in man or animals, but probably virulence is not greatly impaired.

Feeding experiments with true Gaertner group bacilli are by no means so uniform in their results as inoculation experiments, even when bacilli directly isolated from the animal body, and the cause of definite outbreaks of food-poisoning, are employed.

Feeding experiments sometimes produce severe infection, with death, sometimes slight infection, with recovery, and not infrequently no observable symptoms at all.

The bacilli isolated from the Wrexham, St. Anne's-on-the-Sea, and Accrington outbreaks, when used to feed mice or guinea-pigs, caused infection and death, but with the Murrow and Limerick bacilli feeding experiments were negative. In the Limerick outbreak, although the *B. enteritidis* isolated was very virulent to laboratory animals when injected, guinea-pigs fed with cultures remained alive. McWeeney also failed to infect a dog by feeding with a large quantity of meat upon which the bacillus had been grown in the incubator. The dog, which had been kept hungry, eagerly devoured the meat without the slightest ill-effects.

In the Chesterfield outbreak, due to *B. suipestifer*, no feeding experiments were carried out with pure cultures, but four mice and four guinea-pigs were fed with some of the organs of a fatal human

case. Three of the mice died within five days, and one guinea-pig in four (?14) days. In two of the British outbreaks unintentional experiments with domestic animals were made. In the Partick outbreak (No. 34), in one house the cat ate a good deal of the meat. The animal suffered from extreme sickness, vomiting and purging, and continued after the subsidence of the acute symptoms in a lethargic state for several days. At Tollesbury (No. 42) a dog ate some of the infected brawn (5 days old), and shortly afterwards was taken "queer," lying about and refusing its food, but it was given castor oil and soon recovered.

A very important consideration in relation to food-poisoning is the resistance of members of this group to heat. They all behave alike, and being non-sporing bacilli are fairly easily killed, a comparatively short exposure (30 minutes) at 60° C. being sufficient to ensure their destruction. Their toxins, on the other hand, are characteristically heat-resistant. This was first shown by Gaertner with the original *B. enteritidis* isolated by him, and has since been confirmed by very numerous investigators. Van Ermengem, for example, found the toxins of his Moorseele bacillus (*B. enteritidis*) to resist heating to 100° C. or even up to 120° C. Cathcart found that both *B. enteritidis* and *B. paratyphosus B* toxins withstood heating to 100° C. for thirty minutes.

As shown by Gaertner, Fisher, Holst and others, the production of heat-resisting toxins is a property which rapidly becomes lost when the bacilli are artificially cultivated outside the animal body.

In many of the recorded outbreaks shown to be due to the ingestion of Gaertner group bacilli or their toxins, the food had been cooked, while the evidence showed that infection must have taken place before cooking. These cases can be readily explained. On the one hand their heat-resisting properties prevent any pre-formed toxins being destroyed by ordinary cooking, while even the bacilli themselves, embedded as they may be in pieces of meat or fat, are not readily destroyed. On the other hand, much of the cooking to which the food is subjected is very incomplete, and the temperature attained is not high. Meat is a poor conductor of heat. Even with roasted meat, Vallin showed that the temperature of the centre may not go beyond 50° C., or even sometimes 45° C. Wohlflügel and Hüeppe found that it was necessary to prolong the cooking of a leg of veal for $3\frac{1}{2}$ hours at 101° C. for the temperature of the deep parts to reach 71°–89° C.

Petri also tested the penetration of heat into large pieces of meat and found that even after $3\frac{1}{2}$ hours' cooking the temperature of the interior may be only 84° C., or less.

With made foods the temperature reached may be still lower.

In connection with the Derby outbreak, Delépine and Howarth [57] carried out some experiments upon the temperature reached in baking. They noted—

(*a*) The temperature of the centre of a pie said to be under-baked, but having all the external appearances of being well baked, may not exceed 47·2° C.

(*b*) The temperature of the centre of a pie obviously over-baked, and acknowledged to be so, had not got beyond 86·6° C.

(*c*) There was a difference of several degrees between the temperature of various pies.

As Delépine points out, a batch of pies prepared in a hurry might be so cooked that bacteria might continue to grow in their centre during the greater part of their stay in the oven, and the bacteria would certainly not be killed.

In the Murrow outbreak the brawn was certainly infected before preparation, but the temperature reached in the method of cooking used was insufficient to kill the Gaertner bacilli.

I carried out the following experiment in 1908 ([111]). Three sausages were purchased and all three inoculated with *B. suipestifer* (recently isolated from the Murrow outbreak). After inoculation the sausages were incubated for about twenty hours, so that the infection would be massive. One sausage was kept as a control, the other two were fried in a frying-pan in the ordinary way, one being lightly cooked and the other well cooked. All three sausages were then bacteriologically examined, and the Gaertner bacillus was very readily isolated from all of them.

In a number of the outbreaks included in this report the illness caused was clearly due to toxins of Gaertner group bacilli contained in tinned meats. In these cases the cooking had been insufficient to render the meat harmless. In this connection the experiments of Beveridge and Fawcus ([17]) upon the penetration of heat into the substance of meat in tins are of considerable interest.

They found that when a tin was simply boiled in water, the temperature of the centre of the meat did not reach 100° C. even at the end of five hours.

With higher temperatures the following results were recorded :—

Outside Temperature.	Size of Tin.	Time taken by Central Thermometer to		Number of Experiments.
		Reach 100° C.	Reach 105° C.	
107° C.	1 lb.	58 min.	80 min.	Average of 5
107° C.	2 lbs.	95 „	123 „	„
120° C.	1 lb.	22 „	27·4 „	„ 5
120° C.	2 lbs.	28 „	36·2 „	„ 5
130° C.	2 lbs.	17 „	22 „	„ 2

With large 7 lb. tins the rate and time of heat penetration would be much longer.

These experiments illustrate the ease with which the toxins of Gaertner group bacilli may escape destruction in tins of meat insufficiently heated.

The ordinary methods of preservation such as smoking and pickling in brine are not very efficacious to destroy Gaertner group bacilli.

In a series of experiments with commercial brines and artificial salt solutions ([113]), I showed that a rapid elimination of both Gaertner and Coli bacilli took place, at all the temperatures tested, in solutions containing 15 per cent. or more of salt. In 10 per cent. salt solutions the death of the bacilli was also rapid, but less so than

for more concentrated solutions. In one experiment, however, with this strength of salt the bacilli persisted for as long as 28 and 38 days respectively.

SECTION VI.

Sources and Methods of Infection in Food Poisoning Outbreaks.

Previous sections have dealt with various facts ascertained from a study of individual outbreaks and from the bacteriology of the Gaertner group. In the present section the paths of infection and the nature of the sources of infection will be studied.

It has been shown that as bacteriological investigations of outbreaks become more exact and complete, Gaertner group bacilli are isolated in an increased proportion of instances, and while it is possible that individual cases and limited outbreaks of illness may be due to putrefactive changes in the food consumed, it may be accepted generally that infection of food with bacilli of the Gaertner group must be looked upon as the essential cause of outbreaks of food poisoning.

Consideration of the origin of these bacilli and the way or ways they may gain access to the food is therefore of primary importance in discussing sources of infection.

It is unfortunate that so many of the British recorded outbreaks are lacking in information in regard to the paths of infection. Many of the recorders of these outbreaks seem to have deemed it sufficient to prove the possibility of excretal contamination of the food material in question, and having done so appear to have regarded this as a sufficient explanation of the whole occurrence. A few, however, of the records contain information of the highest value, while many of the Continental recorded outbreaks have been more carefully worked out from this point of view. Two further considerations require to be dealt with.

Evidence as to Disease in the Animals supplying the Food Affected.

The evidence as to the health of the animals supplying the meat in the reports dealing with individual outbreaks in this country is very meagre, the majority making no mention of it. In a few cases the investigator states that he was informed that the animals were healthy, but as this is usually the evidence of highly interested persons it is not very valuable.

In only two of the British outbreaks is there any evidence given that the animal was diseased, *i.e.*, No. 43, Murrow, and No. 53, Limerick. In the Bakewell outbreak, No. 48, the persons affected had received their milk from a farm upon which a cow had recently calved, but no evidence of any causal relationship was proved.

In the Murrow outbreak, amongst the bones, &c. used to make the brawn was a pig's foot which was obviously diseased and which

from the description available probably had an abscess on it. The pig was sufficiently affected to have to be carted to the place of slaughter.

In the Limerick outbreak the available evidence is not very precise, but the meat was purchased *ready killed* by the contractor and at *an unusually low price.* No reliable information was obtainable as to the condition of the animal prior to or at the time of slaughter, but since the butcher who sold it to the contractor would appear to have sold it below cost price it is highly probable that it was not sound healthy meat.

In striking contrast are the Continental records. In nearly all the earlier recorded outbreaks, and in a large proportion of the total number recorded, it has been found that the meat was derived from a diseased animal. The Continental outbreaks recorded in Table II. serve as a good illustration. Excluding the two cases not due to animal food and the two instances of fish poisoning the remaining 40 gave the following :—

Animals showing definite signs of disease ...	20
No information given or no disease found ...	20

The diseased conditions found were :—

Gastro-enteritis, &c.	8
Udder disease (2) or puerperal sepsis (1) ...	3
Local abscesses	3
Other diseases	6

In 50 per cent. of the Continental outbreaks (Table II.) compared with not more than 5 per cent. of the British outbreaks (Table I.) definite disease of the animal supplying the food was found. In explanation of this wide discrepancy several points may be mentioned.

(*a*) The two sets of outbreaks are not altogether comparable, since the Continental are in the main large well defined outbreaks, while the British include a number of cases confined to one or two families. Since, however, many of the British outbreaks were also extensive this can only be a partial explanation.

(*b*) Most of the recorded Continental outbreaks were in Germany or Belgium. In those countries the system of meat inspection is more highly organised than in England, and diseased conditions in the animal are therefore more readily traced than in this country, where such conditions are not infrequently overlooked and are rarely recorded.

In these circumstances it is always difficult to obtain information as to the health of the animals supplying any particular specimen of food, and when the food vendors have supplied an article which has caused disease amongst its consumers, this difficulty is not likely to be lessened as the information supplied might incriminate themselves. Also, in addition to the possibilities of wilful suppression of facts, it is very likely that in the absence of skilled supervision, conditions of disease which may be very important, but which are without obtrusive physical signs, would be overlooked.

In illustration of the difficulties in the way of obtaining accurate information it may be mentioned that in the Murrow outbreak quoted above it was only with the greatest difficulty and perseverance that information could be obtained as to the condition of the pig. If thorough and persistent inquiry had not been made this outbreak also would have been returned as one in which no information was available as to the health of the animal furnishing the noxious meat. It is a reasonable inference that if a more systematic inspection of food were in vogue in this country more outbreaks of food-poisoning would be linked up with definite disease in the animals supplying the food.

(*c*) It may be contended that the conditions found in the animals are concomitant lesions, only ascertained because of the rigid inspection and that these lesions are really common conditions and have nothing to do with the disease-producing properties of the meat.

While this contention is one which should be borne in mind, and in favour of which there is some evidence, it cannot be accepted as an explanation of the differences found. In its favour it may be noted that in certain of the outbreaks, for example, No. 110 and No. 114, the lesions found were slight (see Table II), while in the Rohrsdorf outbreak (No. 82) the bacilli were not isolated from the abscesses.

On the other hand, we have the undoubted fact that in a number of these outbreaks abroad a true Gaertner group bacillus was isolated not only from the infective meat and from the patients who died or were made ill from its consumption, but also from the internal organs of the animals supplying it.

In the Cotta (No. 81), Moorseele (No. 84), Aertryck (No. 93) and Meirelbeek (No. 95) outbreaks the *B. enteritidis* or *B. suipestifer* found were isolated not only from the meat or the fatal cases, but also, in each instance, from the marrow of the diseased animals. In the outbreak described by Goebel in Flanders the horse supplying the meat causing the disease suffered from gastro-enteritis, and in addition had an epigastric abscess. *B. suipestifer* was isolated by Goebel not only from the meat and from the fatal case, but also from the pus of the abscess.

Evidence pointing to Infection of the Food subsequent to Slaughter.

A study of recorded outbreaks in this country shows, in a considerable number of them, that infection of the foodstuff may with great probability be accepted as taking place during or after preparation of the animal for sale. The grounds for this view are the following :—

(*a*) *That no information was available showing that the animal was diseased, while clear evidence was forthcoming that during preparation extensive excretal contamination was possible and likely.*

The value of the negative evidence as to the healthiness of the animals has been already dealt with.

The possibilities of excretal contamination were shown to be very great in many of the outbreaks. Three examples out of many may be

mentioned. In the Mansfield outbreak (No. 17) the potted meat, the consumption of which caused the outbreak, was put to set and cool in the preparing-room, which was used for slaughtering the pigs. Pigs had been slaughtered there the day before the incriminated food was made. Further, the utensils used for preparing the potted meat were used equally in connection with the slaughtering. The bowl used for ladling the gravy of the potted meat was, for example, used in dressing the carcases.

In the Wigan outbreak (No. 37), due to potted tongue, the tongues were first put into brine, but the evidence is against the brine being at fault. After removal from the brine the tongues were boiled, and there is no evidence that this was not well done. After boiling, the tongues were put into the vessels, and jelly, whilst hot, poured on to them. The tins of tongue were then put to cool on the *floor* of the slaughter-house, near the steps leading down into the cellar. Since both pig and beef tongues were infected, it is highly probable that both were originally sound, and were infected after the jelly was poured in. The jelly could not have been infected when poured on, since the pigs' heads which received the same jelly were not infected. It is noteworthy that the vessels with the pigs' heads were left to cool in a portion of the slaughter-house which was partitioned off (containing the gas engine).

In the Bacup (No. 79) outbreak the vehicle of infection was roast leg of pork. The legs were stuffed, roasted and sold as boneless legs of pork, so that considerable manipulation was required. The other parts of the pigs caused no illness, and, as it was the legs which were infected, it is reasonable to assume that infection was post-slaughter. An obvious means of possible contamination was found, since it was ascertained that the man who prepared the legs for roasting and removed the bones was also engaged in slaughtering and in *gut cleaning*.

On the other hand, the possibilities of the occurrence of excretal contamination cannot be accepted, apart from other evidence, as sufficient proof that excretal contamination *per se* was the cause. Indeed, any such view is weakened by the fact that such possibilities of contamination exist in a large proportion of the premises throughout the country where food of this character is prepared. It is, for example, a very common practice to put brawn to cool on the floors of rooms liable to considerable contamination from dust. Again, sausages are often prepared, and the pickling of meat is often carried out, in slaughter-houses where excretal material abounds.

In a few of the outbreaks the conditions found have been well above the average, *e.g.*, Accrington (No. 33).

(*b*) *That in many cases the meat during preparation was so treated*, e.g., *by boiling or other form of cooking, that any bacilli or toxins in it must inevitably have been destroyed, and that since the meat was shown to contain Gaertner group bacilli they must have been added after the preparation stages were completed.*

This argument is particularly applicable in the case of brawn and some other prepared foods, and must be considered of great weight. The fact that the food has undergone preparation even involving heating cannot, however, be taken as conclusive in itself that the infection was post-slaughter. Trade expressions frequently will

not bear scientific interpretations, and boiling to the meat purveyor may not mean a temperature of anything like 100° C., either at any time or over a prolonged period. The low temperatures reached in ordinary cooking have already been considered.

In the Bacup (No. 79) and Murrow outbreaks the roasting and boiling respectively were insufficient to kill the bacilli present, infection in both almost certainly having taken place before cooking. Also, in the recorded tinned meat and salmon outbreaks infection clearly took place before the tins were sealed, so that in these cases the so-called "sterilization" was insufficient to kill the toxins present, and even in some cases the bacilli. Most of the tinned food cases were due to Gaertner toxins, but in the two following living Gaertner group bacilli were present and had successfully resisted "sterilization."

Sheffield (No. 24). A fresh 6-lb. tin of "corned beef" was opened and about 4 lbs. of the meat was sold, chiefly in quarter pounds. A Gaertner bacillus was isolated from it. Since cases occurred on the same day and night after it was opened, and most of the purchasers ate it for their mid-day meal, it is clear that there was no time for it to have become so seriously infected after opening. The very short incubation period shows that much toxin must have been also present and this must have been formed prior to opening. In addition the meat was not quite normal in appearance.

Longton (No. 41). Due to tinned salmon. *B. enteritidis* was isolated from the fatal case and from the salmon. Only a few minutes elapsed between the removal of the salmon from the tin and the ingestion of the dose which caused the fatality. The tin was hermetically sealed and there was no decomposition. It is obvious therefore that the *B. enteritidis* must have been present in the fish before the tin was sealed and subjected to the so-called "sterilization." Here the tin was one containing only a pound of fish so that the heat used must have been very inadequate.

In the Tunstall (No. 38) tinned salmon poisoning outbreak no cultural bacteriological investigations were made, but the history shows that it was clearly due to a living bacillus present in the salmon before the tin was opened.

In the tinned food cases due to Gaertner toxins it is evident that the toxins had resisted the degree of heat to which they were subjected during preparation, and that the food must have been infected with Gaertner group bacilli before canning.

(*c*) *That the specific food which caused the outbreak was non-infectious* (*or at least did not cause infection*) *at first and only became so after the lapse of a definite period of time.*

The following outbreaks are good examples :—

Cressage (No. 26). At a farmhouse three geese were killed and a certain widow helped to prepare and cook them. The giblets were stewed September 26th and eaten by the farmhouse people, while the geese were cooked and eaten the next day (September 27th). Both lots of food were eaten without harm. On September 27th about 2 p.m. the widow took home a portion of the giblets (consisting of gizzard and other parts), the giblets gravy, pieces of goose and slices of beef. These were placed in her pantry at home and eaten cold for supper the same evening and without being recooked.

After the consumption of this meat seven persons were ill and two died. 27 hours elapsed from the cooking of the giblets and their consumption.

Chadderton (No. 21). The veal pies were most virulent on the second and third days after cooking. Those which were eaten on the day of preparation and the first day after cooking seem to have caused little or no ill effects.

Downham (No. 58). The brawn was made May 20th. On May 21st about 11 lbs. were sold and no cases resulted. The rest was sold on May 22nd, 38 ate it and 18 were attacked.

Chesterfield (No. 74). This is of especial interest because every portion of the infected consignment was accounted for. At least 29 persons who ate portions of pies which caused illness in others were not taken ill. Excluding those who took aperient medicines the percentage of sufferers to consumers was—

1st day = 0. 2nd day = 82. 3rd day = 86.
4th „ = 91. 5th „ = 88. 6th „ = 100.
7th „ = 100.

In the Alsfeld outbreak (No. 104) the dried pig's meat was eaten the first day without causing illness, but when it was kept for a day then warmed up and eaten it caused eight cases of illness.

While facts such as these are important, they do not prove that infection took place between the date when the meat was non-infective and when it was infective. In the Chadderton, Wrexham and Chesterfield outbreaks the general facts clearly disprove the view that the absence of or lessened infectivity of the meat in the early period was evidence that infection took place after the first day.

In the Tollesbury outbreak (No. 42) the brawn was clearly infectious throughout, but the following figures were obtained :—

Brawn made September 6th and sold September 6th.
42 ate it September 6th, and 13 were ill=31·3 per cent.
71 „ „ „ 7th, „ 65 „ „ =91·5 „ „
4 „ „ „ 8th, „ 4 „ „ =100 „ „

It would appear that the presence of the toxins favours infection by the bacilli.

Even in the Cressage case, which is of great interest, it by no means follows that infection was subsequent to cooking. The geese may have been diseased and the first cooking sufficient to kill nearly all the Gaertner bacilli, but not quite all, a few surviving. The 27 hours in the warm weather would have given them abundant opportunities to multiply in the rich nutrient material, and so render this food highly infective.

This argument is therefore of value, but is not conclusive in itself.

(d) That since other portions of the animal were eaten without harm the animal itself could not have been diseased, and the cause of the outbreak: inferentially, therefore, the food infection must have been after death.

This argument is adopted in many of the English reports, and while it is not without weight, it is only very partially true. It implies that Gaertner group infections of the food-supplying animals are generalised infections, and that the bacilli are present in all the

organs and parts of the body. This would seem not to be the case, and localised lesions, such as the leg lesion in the Murrow pig, would appear to occur. In the Limerick and Murrow outbreaks, in which disease of the animal was highly probable, the rest of the animals were eaten without apparent harm.

In many of the Continental outbreaks the animals supplying the food were definitely diseased, but while the meat of certain parts of the animal caused disease, consumption of the rest of the flesh was without apparent harm.

The question is probably not merely one of a local versus a general infection, but also one of dosage, the bacilli being more distributed in certain parts of the body, while the cooking processes to which different parts are subjected are not equal.

Several interesting experiments have been carried out bearing upon the presence of Gaertner bacilli in the flesh of diseased or inoculated animals.

Van Ermengem ([40]) fed a calf with 100 cc. of a broth culture of his Moorseele bacillus (*B. enteritidis*). The calf suffered from fœtid diarrhœa with blood in the stools, fever and general illness. The animal was killed and the bacilli demonstrated post-mortem. The flesh of this animal and of an inoculated calf was kept for two days at room temperature, and was then worked up into a meat paste. This was well cooked, but even then caused severe symptoms in animals consuming it.

Poels and Dhont injected a small quantity of a pure culture of the Gaertner bacillus isolated by them into the jugular vein of an ox. The ox was killed 20 minutes later and partially bled out. Bacteriological examination of the organs yielded a few Gaertner colonies from the spleen, liver and blood, but none were found in cultures from the flesh. When, however, a piece of the flesh was kept at 20° C. for 72 hours the bacilli had become abundant, while in the meat kept cold at a temperature not greater than 5° C. a few bacilli only were found. 53 persons ate the meat kept at the low temperature, and of these 15 became ill with headache, gastro-intestinal catarrh, and abdominal pain, the onset being 12–18 hours after eating.

McWeeney injected into the jugular vein of a four months' old calf about 20 c.c. of a broth culture of *B. enteritidis* (isolated from the Limerick outbreak) enriched with the growth of the same bacillus on two agar tubes. The animal was ill within two hours and died seven hours after the injection. The autopsy showed small hæmorrhagic spots on the serous membranes and on the mucous membrane of the stomach. The bacillus was not only recovered from all the organs, but "the meat of the animal was found to be literally swarming with the bacillus, and would no doubt have proved highly virulent if eaten by human beings."

(*e*) *That a large proportion of the outbreaks have been associated with foodstuffs which are very liable to bacterial contamination, and which when so contaminated allow marked multiplication of the added bacteria.*

This is an argument of very great importance and weight. The prevalence of brawn and pork pies as vehicles of infection has already been discussed. Both contain a good deal of jelly and are

admirable nutrient media for bacteria, while both cool slowly and in so doing afford, over a long period, a temperature suitable for very rapid multiplication of Gaertner or other bacilli. While brawn is not invariably sterilized during its preparation it is so in most cases, and as such provides a medium artificially sterilized and freed from competing bacilli, a third factor favouring rapid multiplication. The frequency with which foods containing an excess of gelatinous matter have served as the vehicle in food poisoning outbreaks was first drawn attention to by Ballard ([11]) in his classical report to the Local Government Board in 1890.

A further point in connection with these made-foods is the frequency with which these articles have to be handled, thus increasing the liability to infection. One specific instance will serve as an example.

In the Accrington outbreak (No. 33) Delépine describes carefully how the brawn was made and indicates the possibilities of contamination. In this case the premises were better than the average. The tins used for storing the brawn were dried by being laid upside down on the *floor*. The butchers passing from the slaughter house to the boiling room passed over this floor. The tins used were of such a shape that it was practically impossible for the butchers to lift them when full without one or more of their *fingers* coming in contact with the warm meat.

In the examination of the *jelly* Delépine reported that there were found some shreds of intestinal mucous membrane, although no intestine enters into the composition of the brawn. He was of opinion that in this outbreak the meat was contaminated by the hands of some butcher or butchers who had shortly before handled guts.

Several of the British outbreaks are of particular interest in connection with the place of contamination and the nature of the food.

Clapham (*No.* 76).—Tinned pineapple was purchased on the Saturday, opened on Sunday and made into a jelly by the cook on Sunday morning. It was then kept in the larder for about nine hours when it was placed on the table and eaten at 9 p.m. by eight persons, all of whom were ill and one died. There was a definite incubation period and the severity of the symptoms were not in proportion to the dose, so that it was clearly not a case of metallic poisoning.

No bacteriological investigations were made, but this outbreak was probably due to Gaertner or other bacilli which had infected the jelly and multiplied abundantly in it during the nine hours the food was kept in the larder on a hot July day in 1911. The jelly also would have been hot to start with.

This outbreak is of great interest since there was no question of meat being infected ante-mortem or post-mortem. The presence of a material (jelly) capable of serving as a nutrient medium for the bacteria is here the important thing. The report gives no clue as to how the jelly could have become infected.

Norbiton (*No.* 65).—This is an outbreak in which both the vehicle and cause of infection are left undetermined, but which, after a

careful study of the available data, I am inclined to regard as an example of so-called potato poisoning.

The food eaten consisted of potatoes and fish fried in oil, prepared at a fried-fish shop. The potatoes were the only article eaten in common by *all* the sufferers. The sanitary inspector examined the fish, but does not appear to have examined the potatoes. The potatoes were excluded from being the cause on the ground that the rest of the batch were consumed without harm.

The history of the outbreak can probably be accurately reconstructed from the available data as follows :—

The potatoes as purchased by the fried fish shop keeper were good, since half of them, cooked the evening before, were eaten without harm. The remaining half were washed and scrubbed and left ready for frying on the following day. In this state and without their protective skins, they would furnish an admirable nutrient medium for bacteria (one used daily in many bacteriological laboratories), with which they were probably infected in some way which cannot now be determined. This nutrient medium was kept for at least sixteen to twenty hours in a hot shop (the month was August), so that any added bacteria would have time to multiply enormously. The report states very clearly that it was these potatoes which were fried with the fish and not a fresh batch. Probably the cooking in the oil killed the bacteria and left their toxins, and, indeed, some inoculation experiments carried out with the oil indicated the presence of toxins, which might have been dissolved out from the potatoes. No one appears to have asked or recorded whether the potatoes appeared good in the morning when they were used, and no bacteriological examinations of any of those left over were made. It is, therefore, impossible to indicate the bacteria concerned or to supply conclusive proof that the explanation here furnished is the correct one.

It may be added that the symptoms recorded in this outbreak were exactly similar to those recorded in the outbreaks of potato poisoning on the Continent (by Pfuhl, Dieudonné, Schmiedeberg, &c. Schmiedeberg ([119]), for example, recorded three extensive outbreaks in August, 1892, and July, 1893, involving respectively 357, 90 and 125 men—all soldiers. Very similar symptoms were present in these cases and all recovered.

The characteristic feature common to most of these outbreaks is the fact that usually the potatoes were peeled (and sometimes cooked) the day before they were used. For example, in the outbreak recorded by Pfuhl (56 cases) the potatoes concerned were peeled, placed in a vessel filled with water, and remained standing in the water until next day. They were then washed and cooked for 25 minutes.

The cases of ice-cream poisoning, six of which are included in the series of British outbreaks, may be mentioned here. In these cases it is unlikely that the origin of the bacilli is from milk derived from a diseased cow and the feature which apppears to be of greatest significance is the presence of a food which forms a nutrient medium for bacteria, which is partially sterilized in the making so that the restraining influence of other bacteria is largely ruled out, and which is allowed to cool slowly so that a temperature

suitable for rapid bacterial multiplication is ensured. When it is added that ice-cream is frequently made under conditions which invite infection and that in particular the places where it is put to cool are often most unsatisfactory, it is not to be wondered at that heavy bacterial infection results or that, when specific disease-producing bacteria gain access, food-poisoning outbreaks result.

The food-poisoning outbreaks, in which the vehicle of infection is not of animal nature at all, clearly show that infection of the food during or subsequent to preparation must have taken place.

While no one of the considerations mentioned as evidence of infection subsequent to the death of the animal is, in itself, sufficient to prove the point, they are each of value, and their cumulative evidence is in many cases quite sufficient to prove that the bacteria obtained access to the food after slaughter.

There is one important argument on the other side which has not been mentioned. It is only in extremely rare cases that the sheep has been the animal furnishing the meat which has served as the vehicle of infection. It may be advanced that if post-slaughter infection were common we should meet with many more such cases. since we have no reason to doubt but that Gaertner group bacilli added to mutton will multiply abundantly. The probable explanation is the comparative infrequency with which mutton is used in the preparation of made foods. It is chiefly eaten in the form of roast or boiled meat, and the joints being small are more likely to be efficiently cooked. There are no known diseases of the sheep due to Gaertner group bacilli.

Sources of Infection.

Consideration of the facts set out leads to the general conclusion that in those outbreaks due to infection of the food with bacilli of the Gaertner group (and this probably includes the great majority of outbreaks), the food is contaminated in some cases because it is derived from an animal suffering from a general or local disease caused by one or other member of the true Gaertner group, and in others because, subsequent to slaughter, Gaertner group bacilli from outside sources have gained access to it.

It has been shown in Section V. that diseases of animals used for food, caused by Gaertner infections, are not very uncommon, and their occurrence is sufficient to explain those outbreaks in which definite disease of the animal supplying the meat has been ascertained to have existed. It is at present impossible to state what proportion these outbreaks are to the whole. Probably they will be found to be considerably higher than would be deduced from the British records and considerably lower than the percentage found in the reported continental outbreaks.

In regard to the outbreaks in which the Gaertner group bacilli have gained access from sources outside the meat or food itself, it is an extremely difficult matter accurately to trace their origin, and in explanation three distinct views or hypotheses have been advocated which will require separate consideration.

Hypothesis A. That the Gaertner group bacilli which are the cause of the food-poisoning outbreak are of human origin, the meat being infected with pathogenic Gaertner bacilli from a human source, i.e., *a case of disease (paratyphoid fever) or a carrier case.*

This view has been advanced by Conradi and by Romeller. It has in its favour the fact that paratyphoid bacilli carriers have been proved to exist and that by analogy with typhoid carriers such carriers are potential, and may be actual, sources of infection. There are several recorded outbreaks of food poisoning which may possibly have been due to carriers, for example, the epidemic at Halle described by Baehr and the Wrexham outbreak. In the former the Gaertner bacilli were only found in the excreta of the butcher after the outbreak, although he fell ill shortly before the outbreak, so that it is not possible to settle between the rôle of cause and victim. In the recent outbreak at Wrexham the outbreak has been ascribed to a carrier, but the evidence is very inconclusive. The cook is said not to have eaten any of the incriminated pie, and her blood reacted to the Gaertner group bacillus isolated as the cause of the outbreak in one out of four tests. From her fæces members of the Gaertner group were isolated, but no evidence is advanced that they were identical with the Wrexham bacillus. The cook might have been slightly infected without showing any obtrusive symptoms. All the bacteriological findings were of course after the outbreak.

There are many and weighty objections to this conception of human infection. In the first place it can only serve as an explanation of a limited number of the outbreaks since, in cases of paratyphoid fever, the only Gaertner group organisms present are *B. paratyphosus A* and *B.* While most German authorities claim that *B. paratyphosus B* and *B. suipestifer* are identical, all authorities are agreed that *B. enteritidis* is distinct from either of them, so that this view cannot explain outbreaks due to *B. enteritidis.* Further if, as most English authorities maintain, *B. paratyphosus B* and *B. suipestifer* are distinct organisms, it follows that paratyphoid fever cases or carriers cannot be advanced as the source of outbreaks due to *B. suipestifer.* It has been shown above that in all, or practically all, the food-poisoning outbreaks in this country in which Gaertner group bacilli have been isolated, these bacilli have been either *B. enteritidis* or *B. suipestifer.* Unless, therefore, the identity of *B. suipestifer* and *B. paratyphosus B* is accepted there are no known outbreaks in this country (with one possible exception) for which this conception of a human paratyphoid case or carrier can be advanced as an explanation.

This view does not explain the proved association of many outbreaks with the consumption of the flesh of diseased animals.

It also requires acceptance of the hypothesis, first advanced by Trautmann, that in meat-poisoning outbreaks we have to deal chiefly with an intoxication from ingested toxins, while in paratyphoid fever with an infection with a few of the same bacilli (paratyphoid bacilli), which only cause disease symptoms after a definite incubation period.

If this hypothesis were true, in most outbreaks some cases would

be acute with the ordinary food-poisoning symptoms, while others would be cases of paratyphoid fever. This is by no means the case, although, as explained on page 10, a few protracted cases of infection are met with.

It has been pointed out that in several outbreaks the meat, when undoubtedly infected with Gaertner group bacilli, was eaten on the first day without harm or with only a small percentage of attacks, although very toxic on the second and subsequent days. On this hypothesis, at least, some of the persons who ate the meat on the first day should have developed paratyphoid fever. There is no evidence of a single such case in these outbreaks.

We have evidence that *B. paratyphosus B* can cause acute gastro-enteritis, simulating a food-poisoning outbreak, in the very interesting outbreak recorded by Bainbridge and Dudfield ([10]). They record a number of cases of acute gastro-enteritis in which the onset and symptoms were indistinguishable from those met with in ordinary food-poisoning outbreaks, and in which *B. paratyphosus B* was the organism found. Careful investigation failed to find any article of food which could be shown to be the vehicle of infection while a number of the cases occurred in different places and on different dates. The cases were connected, but it was one of contact, not a common food supply. In this outbreak the evidence was in favour of a human source of infection.

While the above hypothesis may serve as a possible explanation of outbreaks due to *B. paratyphosus B*, it cannot be advanced as a general explanation for all. At present, while we are not in a position to say that food-poisoning outbreaks due to this member of the Gaertner group may not occur, there is no recorded outbreak (at least in this country) in which *B. paratyphosus B* has been proved to be the cause. The Gaertner group bacillus isolated has always been *B. enteritidis* or *B. suipestifer*.

Hypothesis B. That the Gaertner group bacilli which set up the food-poisoning outbreaks are derived from ordinary fæcal infection of the food.

The acceptance of this view, of course, implies that the true Gaertner group bacilli are *natural* intestinal inhabitants. In view of this hypothesis this question was considered in considerable detail in Section V. It was there shown that, apart possibly from rats and mice, true Gaertner group bacilli cannot be considered as natural inhabitants of the animal intestine.

From my own and recorded investigations I believe this hypothesis, which is so frequently given in reports upon food-poisoning outbreaks as a sufficient explanation, is without foundation, and that any support which it has received from bacteriological findings is due to insufficient cultural and agglutinative differentiation of the bacilli isolated.

Also strongly against this view is the fact that present day methods for the preparation of meat for sale very frequently allow great possibilities of contamination with excrementitious matters.

This contamination is especially liable to occur with made-foods such as sausages, brawn and meat pies. My own examinations have shown that the two former frequently show the presence of enormous numbers of fæcal bacilli.

I have also shown that when flesh, brawn, &c. is inoculated with Gaertner bacilli and with *B. coli* both multiply very rapidly, so that there is no reason for assuming that under natural conditions any Gaertner group bacilli which gained access to meat would be overgrown by concomitant *B. coli*, thus preventing infection.

This being the case, if simple fæcal contamination is a sufficient explanation, food-poisoning outbreaks should be extremely common instead of comparatively rare.

The available data are strongly against the correctness of this hypothesis.

Hypothesis C. That food-poisoning outbreaks are due to infection of the food with virulent Gaertner Group organisms (or other special bacilli) derived from animals which are either at the time suffering from disease due to Gaertner Group bacilli or acting as carriers of these bacilli.

In my opinion, this view is one which best explains all or most of the phenomena of food poisoning.

As set out in Section V., there are a considerable number of animal diseases due to Gaertner group bacilli. Many of them affect animals used for human food, others animals (rats and mice) which have opportunities of infecting human food.

This hypothesis is adequate to explain the outbreaks associated with the consumption of the meat of a diseased animal, and is also capable of explaining the outbreaks in which infection occurs during or after preparation of the food.

While true Gaertner group bacilli are not natural inhabitants of the healthy animal intestine, they are very occasionally found to be present in it and in a few cases have been found in made-foods, such as sausages. Their occurrence which is rare is, in my opinion, probably due to the presence of carrier bacilli in animals recovering from infection with a Gaertner group organism.

Zwick and Weichel examined 177 mice and in 28 found they were acting as carriers of Gaertner group bacilli.

Petri and O'Brien ([94]) fed guinea-pigs with *B. suipestifer* cultures. One died. The rest remained apparently healthy during the period of observation, extending over 60 days, and the bacillus used for feeding was isolated from the excreta on a number of occasions. In this way they produced healthy carriers.

O'Brien ([88]) has described an epizootic due to *B. suipestifer* amongst 500 laboratory stock guinea-pigs. All but 21 of them died. Five of the survivors proved to be carriers, excreting the bacilli intermittently five months later. The bacilli were, however, of diminished pathogenicity, while spread of infection from these carrier animals did not take place in the few experiments carried out.

It does not appear to be settled how long these bacilli can retain their virulence when unassociated with a definite pathological condition. From analogy with typhoid carriers, it may be for very long periods. On the other hand, outside the animal body Gaertner group bacilli rather readily lose their heat-resisting properties and to some extent their virulence.

It is probable that the virulence of the infecting bacillus, as well as the number present, is important in initiating infection. Meat-poisoning outbreaks are far more prevalent in the summer months. This is no doubt in part a question of dosage, since the rate of multiplication of these bacilli is greatly favoured by a high temperature. It is, however, possible that in hot weather the virulence of Gaertner bacilli is raised and carrier bacilli, usually of too low virulence to infect, may become definitely pathogenic to man. It must be remembered that while true Gaertner group bacilli are very virulent by subcutaneous or intraperitoneal injection, they are far less virulent by the mouth, the way that human infection in food-poisoning outbreaks is transmitted.

The importance of time in relation to infection is shown in the Mossley (No. 49) outbreak. The beef was used to make three separate batches of meat pies, all made the same day, and only the last batch was poisonous. The meat was all minced about 1 p.m. by machine. The first batch was made at 2 p.m. and the second lot immediately following. The third batch was not made until 6 or 7 p.m. During these five to six hours in August the minced meat was kept in an earthenware mug on a table under an open window. It is, of course, an alternative supposition that the infection only took place after the first two batches were made.

The Gaertner bacilli used for the destruction of rats and mice are evidently of low virulence to man, since very few accidents appear to have occurred when compared with the very extensive facilities offered for infection during handling and in use.

Several groups of cases abroad (for example Schibayama ([118])) and one outbreak in this country have been ascribed to their use. The English outbreak occurred in the City of London and may be briefly described.

In July 1908 an outbreak of illness occurred at a large business establishment where a considerable number of persons of both sexes were employed. Between the 18th and 22nd 12 men became seriously ill, the symptoms being fever, severe headache, vomiting, diarrhœa, cramp in the abdomen, giddiness, with severe collapse in many cases. There were no deaths. The severe symptoms lasted about 48 hours but each case recovered and was practically convalescent at the end of the week.

All those taken ill had their meals in the same dining room and no illness arose amongst those using the other four dining rooms. No article of food was served exclusively in this particular dining room. In the course of the investigation an offensive smell was noticed in the dining room used by the patients, and on removing some of the floor boards on July 30th the bodies, more or less decomposed, of 40 mice were found. It was then ascertained that on July 16th some "Liverpool Virus" rat poison had been put

down in the dining room and in one of the pantries. Only one tube was used and it was only once put down.

Specimens of the stools of some of the patients were bacteriologically examined by Dr. Klein who isolated a bacillus (microbe S.) from the stools, which agglutinated the sera of five of the cases when convalescent. This microbe S. was pathogenic to a guinea-pig and culturally was identical with the Liverpool virus, both organisms in Dr. Klein's opinion showing minute cultural differences from *B. enteritidis*.

It is, however, noteworthy that the sera of the convalescent cases showed only very feeble agglutination reactions with the Liverpool virus or with *B. enteritidis*.

Bainbridge ([6]) has shown that the Liverpool virus is indistinguishable from *B. enteritidis* by cultural, agglutination and absorption tests, so that the failure of the sera of these convalescent cases to agglutinate either the Liverpool virus or *B. enteritidis* throws grave doubts upon the etiological association.

The cultural tests used by Klein were insufficient to distinguish microbe S. from the para-Gaertner bacilli, and it is not clear whether this organism was a para-Gaertner bacillus or a true Gaertner organism such as *B. suipestifer*.

The outbreak is of great interest but the available evidence both on bacteriological and epidemiological grounds is insufficient to prove that it was caused by the rat virus.

It is evident that very much fuller information is desirable in regard to the use of these rat and mice viruses, and particularly as to how far those handling them develop slight infections with the presence of agglutinins in the blood.

SECTION VII.

General Preventive Measures against Food Poisoning.

While recent bacteriological investigations have widened our knowledge considerably as to food poisoning and the distribution of the bacilli concerned, this increased knowledge has shown that the problem of prevention is more complex than was at one time supposed to be the case.

In this it is comparable to the added administrative difficulties which have been caused by our increased knowledge as to the bacteriology and epidemiology of the acute infectious diseases.

It must be admitted that with our present limited knowledge the prevention of food-poisoning outbreaks is one of extreme difficulty.

If the original conception of food poisoning as due to the ingestion of food in a state of incipient decomposition had proved to be correct, prevention would have been a comparatively simple matter,

resolving itself into steps to prevent this incipient decomposition and a careful instruction of the public as to the evil effects of tainted meat.

If the hypothesis which followed it—that these outbreaks were entirely due to the consumption of the meat of animals suffering from certain specified diseases—had been shown to embrace the whole truth as to food poisoning, satisfactory preventive measures might have been devised, consisting in a more rigid inspection and supervision of all meat at the time of slaughter.

It, has, however, been shown in the earlier sections that the first hypothesis is not in accord with modern knowledge, while the second does not account for all the cases.

From the review of existing knowledge given in the foregoing sections it is clear that, from the preventive standpoint, food poisoning cases must be looked upon as forming two groups :—

(*a*) Those due to the consumption of meat derived from diseased animals; an *intra-vitam* infection with food-poisoning bacilli, in most cases members of the Gaertner group, but probably occasionally with other varieties of bacteria.

(*b*) Those due to the contamination of healthy meat or other food with food-poisoning bacilli derived from sources other than the food itself.

While available knowledge is insufficient to enable all sources of contamination to be defined with accuracy, it is probable that the second group is considerably larger than the first.

Dealing with the second group, preventive measures must necessarily be framed on the basis of the hypothesis which is most in accordance with the facts, viz., that the infecting Gaertner group or other bacilli are in most of the cases in this group, if not in all, derived from the intestinal contents of animals (either the domestic animals used for food or rats and mice) which are themselves suffering from infections caused by these bacilli or which are acting as carriers of the bacilli.

In view of the above, the following preventive measures require careful consideration.

1. *Meat Inspection at the time of Slaughter.*

The extent to which a more rigid supervision of the slaughter of animals used for human food is likely to prevent the incidence of food poisoning must be considered.

In outbreaks due to the consumption of the flesh or organs of diseased animals most of these animals would be detected by an efficient system of meat inspection, and such outbreaks would, to a large extent, be prevented. Even with such a system, however, outbreaks from this cause would probably still occur, as in some cases the disease (enteritis, &c.) from which the animal was suffering might be overlooked. In several of the recorded continental outbreaks the meat was passed by the veterinary meat inspector although it was subsequently ascertained that small lesions were present which were probably associated with the presence of the Gaertner group bacillus causing the outbreak.

There is, indeed, sometimes considerable difficulty in ascertaining how far Gaertner bacilli are likely to be present in some of the diseased conditions in animals associated with food poisoning. In Germany the provision of bacteriological laboratories in connection with public abattoirs has been advocated and in some cases provided. While bacteriological facilities should certainly be available in doubtful cases in connection with meat inspection it is obviously impracticable to use bacteriology for routine work.

Under the system of isolated slaughterhouses in vogue in this country it is impossible to exert any adequate supervision over the slaughter of animals for food, and it is easy for diseased animals infected with Gaertner bacilli to be put upon the market and sold as healthy. No additional bacteriological facilities would in such circumstances prevent this.

2. *Separation of the slaughterhouse from the food-preparing places.*

A careful study of the facts of individual outbreaks as well as of theoretical considerations, shows that the separation of the slaughterhouse from the place where meat is cut up and prepared for food is a most important preventive measure. In addition to the actual slaughtering, secondary procedures are frequently carried out in the slaughterhouse. The guts of pigs and other animals are not infrequently cleaned and washed in the slaughterhouse and then put in salt solution and used for sausage-casings. Brawn is also sometimes made and is frequently set to cool in the slaughterhouse, and it is not at all uncommon for sausages to be made and for the vessels containing the pickling fluid and meat in course of salting to be kept in the slaughterhouse.

It is obvious that these practices invite contamination of the food and, if a diseased animal is being handled, may cause specific excretal infection of the food and consequent liability to outbreaks of food-poisoning.

3. *Cleanliness in premises where made-up meat foods are prepared.*

The most striking fact which emerges from the study of any series of meat poisoning outbreaks is the very large number of cases in which the vehicle of infection has been brawn, meat pies, or other form of made-up meat. Investigation of the condition of the premises and the actual procedure practised has almost invariably shown that, speaking generally, cleanliness was not practised in the preparation of these materials nor were the premises of such a kind as would ensure cleanliness and freedom from contamination. Definite opportunities for excretal contamination of the food, during preparation or subsequently, were usually present.

4. *The Limitation of Diseases in Animals caused by Bacilli of the Gaertner group.*

Looking beyond the immediate steps required to prevent the infection of food for human consumption, an important practical

inference follows from the hypothesis that the majority of food-poisoning outbreaks are due to bacilli of the Gaertner group and that (as explained in Section VI.) these bacilli are derived from animals themselves specifically infected with these organisms ; namely, that any steps which can be taken to limit the prevalence of disease in animals due to bacilli of the Gaertner group will diminish the risk of specific infection of human food and so will tend to lessen the number of outbreaks of food-poisoning.

Such limitation of the amount of Gaertner disease in animals would be difficult to secure and its detailed discussion is beyond the scope of the present report. In this connection arises the question as to the advisability of allowing the unchecked distribution of bacilli of the Gaertner group in the form of the various rat and mice viruses, for the purpose of destroying these rodents. These bacilli differ only in virulence from the Gaertner group bacilli associated with food-poisoning outbreaks, and although they are stated to be non-pathogenic to man (and numerous instances of their free use without harm favour this contention), there are several instances in which cases or groups of cases of illness in man have been attributed to infection from these viruses.

One prominent characteristic of the Gaertner group of organisms is their varying virulence, and we are not in a position to negative the possibility that strains, originally non-virulent to man, may, by single or repeated passage through rats or mice, acquire a virulence sufficient to enable them to infect man, either directly, or indirectly by causing disease in the animals used for human consumption.

The danger of contamination of food by excreta of rats has been dealt with in Section VI., p. 55.

5. Steps to obtain Official Cognisance of Food-poisoning Outbreaks.

There is not the slightest doubt that a very large number of cases, and even considerable outbreaks of food-poisoning and food infection, never come to the notice of the official authorities, and remain unrecorded, uninvestigated and sometimes unrecognised. As illustrating how slender the chances may be of such cases coming to light the Bristol outbreak (No. 56), recorded in Table I., may be quoted. No less than 54 known cases are recorded, but all of them were ill four days before the outbreak came to the notice of the health authorities. This information was purely fortuitous as it was only obtained through one of the office staff reporting that his wife and neighbours were suffering. It is highly probable that if an employee had not been affected the health department would have known nothing about it.

If adequate measures are to be taken to prevent the recurrence of illness due to food-poisoning, it is essential that all outbreaks, however small, should be brought to the knowledge of the medical officer of health and be thoroughly studied by him, with the assistance at an early stage of a bacteriologist.

The memorandum of the Local Government Board dated September, 1911, on the investigation of outbreaks of illness

suspected to be due to food-poisoning indicates the lines on which such enquiries should be conducted and will, no doubt, do much to stimulate interest and to ensure investigation of all such outbreaks which are brought to the notice of the medical officer of health. This memorandum is reproduced as Addendum B., p. 68.

W. G. SAVAGE.

A. W. J. MacFadden,
Chief Inspector of Foods,
Local Government Board,
July, 1913.

ADDENDUM A.

Table I.—British Outbreaks.

Number.	Place.	Year.	Month.	Incubation period (in hours).	Number of Cases.	Case infectivity (where known).	Deaths.	Kind of food eaten.	Animal from which derived.	Bacteria isolated.
1	Whitchurch ...	1878	—	4–19	—	—	2	Roast leg of pork ...	Pig ...	—
2	Welbeck ...	1880	June ...	12–36 for most	72+	—	4	Cold ham	Pig ...	The Welbeck bacillus.
3	Nottingham ...	1881	February	7–24	15	—	1	Hot baked pork ...	Pig ...	—
4	Oldham ...	1882	—	½–5	9	100 per cent.	0	Tinned tongues ...	Pig ...	No bacteria isolated.
5	Bishop Stortford	1882	—	—	3 families	—	0	Ribs of beef	Ox ...	—
6	Whitchurch ...	1882	—	1–5	10 families	—	0	Brawn	Pig ...	—
7	Wolverhampton	1884	May ...	10–14	3	100 per cent.	2	Tinned salmon ...	Salmon	—
8	Carlisle ...	1886	August ...	6–43	20	83 per cent.	1	—	—	—
9	Iron Bridge ...	—	—	6–12	12	—	0	Veal pies	Calf ...	—
10	Retford ...	1887	November	6–30	80	—	1	Pork pies and brawn	Pig ...	—
11	Carlisle ...	1889	November	about 24	25	—	0	Pork pies and boiled salt pork.	Pig ...	A bacillus.
12	Portsmouth ...	1890	February	14–17	12	96 per cent.	0	Meat pies	—	—
13	London ...	1889	March ...	about 24	8	—	0	—	Fish ...	—
14	London ...	1889	May ...	about 11	4	—	0	Pork	Pig ...	—
15	London ...	1889	August ...	about 5	5	—	1	Beef	Ox ...	—
16	London ...	1895	June ...	About 12	3	—	0	Meat pies	Ox ...	—
17	Mansfield ...	1896	February	12–24 hrs. for most.	265	95 per cent.	0	Potted meat	Ox and Pig.	(Inconclusive).
18	Atherton ...	1898	—	1–2	—	—	—	Pickled tongues ...	Ox ...	—
19	Hatton ...	1898	—	—	185	—	1	Veal pies	Calf ...	*B. suipestifer.*
20	Salford ...	1898	June ...	About 6–12	5	100 per cent.	1	Cold hashed beef ...	Ox ...	*B. suipestifer* (from serum reactions).

Table I.—British Outbreaks—*continued.*

Number.	Place.	Year.	Month.	Incubation period (in hours).	Number of Cases.	Case infectivity (where known).	Deaths.	Kind of food eaten.	Animal from which derived.	Bacteria isolated.
21	Chadderton(and Oldham).	1898	July ...	Most 6–7; 3–29 as extremes.	47	—	4	Veal pies	Calf ...	*B. suipestifer*
22	Surbiton ...	1898	August ...	8–44	10	—	2	Fresh ox tongue (also ribs of lamb).	Ox ...	*B. suipestifer.*
23	Nuneaton ...	1899	April ...	5½–48	42	Apparently 100 per cent.	0	Chitterlings	Pig ...	—
24	Sheffield ...	1899	November	1–3½	24	100 per cent.	1	Corned beef	Ox ...	*B. enteritidis.*
25	Rochdale ...	1900	August ...	—	141	—	0	Ice cream	—	—
26	Cressage(Salop)	1900	September	3–5	7	100 per cent.	2	Goose gravy and giblets	Goose	—
27	London (Islington)	1901	July ...	—	4	100 per cent.	1	Cream (in jar) ...	—	—
28	London (City)	1902	July ...	Within a few hours.	17	—	0	Ice cream	—	—
29	London (Fulham)	1902	September	—	4	100 per cent.	1	Stewed rabbit, warmed up.	Rabbit	*B. suipestifer.*
30	Derby	1902	September	4–24	221+	—	1	Pork pies	Pig ...	Gaertner.*
31	Battersea ...	1904	July ...	8–14	64+	—	0	Ice cream	—	(Inconclusive)
32	Birmingham ...	1905	July ...	½–8½	52	About 20 per cent.	0	Ice cream	—	(Inconclusive)
33	Accrington ...	1906	Jan.–Feb.	12–36	50	—	0	Brawn...	Pig ...	Gaertner.
34	Partick ...	1906	May ...	1–4	12	—	0	Tinned beef	Ox ...	Gaertner (from serum reaction).
35	Sculcoates Workhouse.	1906	June ..	—	35	—	6	Frozen meat	—	—
36	Bedford ...	1906	June ...	8–16	29	—	0	Pork pie	Pig ...	Gaertner.
37	Wigan ...	1907	January	—	168+	—	0	Potted tongues ...	Ox and pig.	Gaertner.

* Available information insufficient to state the precise Gaertner sub-group. This applies to each instance when "Gaertner" is used in the Tables.

38	Tunstall ...	1907	April ...	10–14	3	37·5 per cent.	1	Tinned salmon ...	Salmon	—
39	Partick ...	1907	June ...	About 24	3	100 per cent.	0	"Hough and potted chicken."	—	*B. enteritidis.*
40	Ashton-under-Lyne.	1907	August ...	4–17	44+	—	0	Udder	Cow ...	—
41	Longton ...	1907	August ...	12–24	4	100 per cent.	1	Tinned salmon ...	Salmon	*B. enteritidis.*
42	Tollesbury ...	1907	September	1½–4	80	—	0	Brawn	Pig ...	*B. enteritidis*
43	Murrow ...	1908	May ...	12–48	18	100 per cent.	3	Brawn	Pig ...	*B. suipestifer.*
44	St. Annes-on-the-Sea.	1908	July ...	2–12	76	95 per cent.	3	Potted beef (34 cases) Pork pies (42 cases)	Ox ... Pig ...	Gaertner.
45	Whitefield ...	1908	July ...	—	11	—	0	Pork pies	Pig ...	—
46	Newcastle-under-Lyme.	1908	July ...	6–12	31	—	0	Milk	Cow ...	*B. Coli.*
47	Hornsey ...	1908	July ...	—	6	75 per cent.	1	—	Shrimps	Gaertner.
48	Bakewell ...	1908	August ...	—	30+	—	0	Milk	Cow ...	—
49	Mossley ...	1908	August ...	1–16	7	—	2	Beef pies	Ox ...	Gaertner.
50	Blackpool ...	1908	September	—	29	About 25 per cent.	0	Pork pies	Pig ...	—
51	Wirksworth ...	1908	September	—	—	—	1	Beef brawn	Ox ...	Gaertner.
52	Disley and Marple.	1908	October...	—	About 50	—	1	Brawn...	Cow ...	—
53	Limerick ...	1909	May ...	—	73	—	9	Beef	Ox ...	*B. enteritidis.*
54	Nuneaton ...	1909	June ...	3–9	67	—	0	Ice-cream	—	Gaertner.
55	London ...	1909	July ...	8–13	7	—	0	Steak and kidney pie	Ox ...	*B. Coli*, &c. (no Gaertner).
56	Bristol ...	1909	August ...	—	54+	83 per cent.	0	Corned beef	Ox ...	—
57	Kensington ...	1910	April ...	Within 12 hrs.	8	—	0	Sausages (from the sausage casings).	—	(Inconclusive).
58	Downham ...	1910	May ...	1–5	18+	—	0	Brawn	Pig ...	—
59	Warwick ...	1910	June ...	20 or more for most.	20+	—	1	Boiled (fresh) beef ...	Ox ...	Gaertner.
60	St. Helens ...	1910	August ...	2–10 (3–6 hrs. for most).	32	97 per cent.	0	Tinned corned beef ...	Ox ...	Inconclusive.
61	Astley	1910	August ...	Within 1 hour	7	100 per cent.	0	Tinned tongue ...	?	—
62	Burslem ...	1910	August ...	7–8	4	—	1	Tinned salmon ...	Salmon	—

TABLE I.—BRITISH OUTBREAKS—*continued.*

Number.	Place.	Year.	Month.	Incubation period (in hours).	Number of Cases.	Case infectivity (where known).	Deaths.	Kind of food eaten.	Animal from which derived.	Bacteria isolated.
63	Castle Bromwich.	1910	August ...	—	12	About 24 per cent.	1	Ice-cream	—	—
64	Wrexham ...	1910	August ...	5–45 (12 hrs. for most).	107	—	5	Pork pies	Pig ...	*B. paratyphosus B* (*B. suipestifer*)
65	Norbiton ...	1910	August ...	All within ½ hour.	80–100	—	0	Fish, potatoes, oil ...	—	(Inconclusive).
66	Stretford ...	1910	September	—	20	—	0	Braised beef	Ox ...	—
67	Rowley Regis...	1910	October...	—	20–30	100 per cent.	0	Cold boiled beef ...	Ox ...	Inconclusive.
68	Ramsgate ...	1910	October...	—	30+	—	0	Cold boiled ham ...	Pig ...	Inconclusive.
69	Saltley (Birmingham).	1910	December	1–3	4	100 per cent.	1	Sausage rolls	—	—
70	Bingham ...	1910	—	—	—	—	0	Kippers	Herring	—
71	Chester ...	1911	March ...	½–6	10	—	0	Brawn...	Pig ...	—
72	Warrington ...	1911	April ...	9–16	23	96 per cent.	0	Corned beef	Ox ...	*B. enteritidis* (from serum reactions).
73	Edinburgh ...	1911	June ...	—	About 100	—	—	Brawn...	Pig ...	Inconclusive. No Gaertner.
74	Chesterfield ...	1911	June ...	—	168	—	1	Milk	Cow ...	Gaertner.
75	Bacup... ...	1911	June ...	4–20	213+	—	4	Pork pies	Pig ...	*B. suipestifer*.
76	Clapham ...	1911	July ...	6–9	8	100 per cent.	1	Brawn, roast pork (from same pigs).	Pig ...	Gaertner.
77	Walthamstow	1911	September	—	7+	—	1	Pineapple jelly ...	—	—
78	Wigan... ...	1911	September	2	11	100 per cent.	0	Tinned tongue ...	—	—
79	Bacup ...	1911	October...	—	38	90 per cent.	0	Corned beef	Ox ...	—
								Roast leg of pork ...	Pig ...	(Inconclusive).

Table II.—Continental Outbreaks.

Number.	Place.	Year.	Month.	Number of Cases.	Deaths.	Kind of food eaten.	Animal from which derived.	Bacteria isolated.	Information as to disease of animal supplying the food, remarks, &c.
80	Frankenhausen	1888	May ...	57	1	Meat	Cow	*B. enteritidis*	Cow suffering from gastro-enteritis.
81	Cotta	1889	June ...	126	4	Meat	Cow	*B. enteritidis*	Cow emergency slaughtered suffering from purulent inflammation of the udder.
82	Rohrsdorf ...	1890	—	—	—	Sausages from liver and flesh.	Horse	*B. enteritidis*	The horse had suffered from abscesses but the Gaertner bacillus was not isolated from them.
83	Gaustad ...	1891	June ...	81	4	Baked meat ...	Calf...	Gaertner* ...	Calf suffered from enteritis 14 days before its slaughter.
84	Moorseele ...	1891	August ...	80	4	Veal...	Calf...	*B. enteritidis*	Meat from two calves suffering from severe enteritis. One died the other was killed when on the point of death.
85	Rotterdam ...	1892	—	92	0	Meat	Cow	Gaertner ...	Cow inspected in the ordinary way and passed as healthy.
86	Rumfleth ...	1893	September	19	0	Meat	Cow	Gaertner ...	Cow fell ill after calving and died.
87	Breslau ...	1893	October...	80+	0	Meat (minced) ...	Cow	*B. suipestifer*†	Cow killed on account of diarrhœa and fever.
88	Bischofswerda	1894	May ...	70–100	0	Pork sausages; chopped beef.	Pig and Cow.	Gaertner ...	Food eaten raw. No information as to illness of the animals.
89	Haustedt	1895	June ...	50	0	Meat	Ox ...	*B. enteritidis*	Cow emergency slaughtered on account of diarrhœa and fever.
90	Ghent	1895	October...	12	1	Raw saveloy ...	—	*B. enteritidis*	—
91	Horb ...	1896	September	50	0	Liver sausage and meat.	Calf...	*B. enteritidis*	Calf 5–6 weeks old and suffered from arthritis.
92	Posen	1896	—	26–27 families.	1	Pork	Pig ...	*B. suipestifer*	No evidence that the animal was diseased.
93	Aertryck ...	1898	July ...	—	1	Veal...	Calf...	*B. suipestifer*	Calf suffered from severe enteritis.

* *See* footnote to Table I.

† *B. suipestifer* and *B. paratyphosus B* are accepted as identical in most of the German records, the organisms isolated from outbreaks being sometimes called the one, sometimes the other bacillus. With the nomenclature used in this Report all these organisms are probably *B. suipestifer* and are here so recorded.

Table II.—Continental Outbreaks—*continued.*

Number.	Place.	Year.	Month.	Number of Cases.	Deaths.	Kind of food eaten.	Animal from which derived.	Bacteria isolated.	Information as to disease of animal supplying the food, remarks, &c.
94	Sirault ...	1898	August ...	100+	3	Chopped meat ...	Pig ...	*B. suipestifer*	—
95	Meirelbeek ...	1899	—	—	—	Meat	Cow	*B. suipestifer*	Animal emergency slaughtered for puerperal sepsis.
96	Düsseldorf ...	1901	November	57	1	Chopped meat ...	Horse	*B. suipestifer*	In all but four cases meat eaten raw. No evidence of disease amongst the animals.
97	Neunkirchen ...	1903	May ...	50+	3	Meat sausage ...	Horse	*B. enteritidis*	Animal suffered from abscesses.
98	Grotz-Barnitz	1903	November	19	—	Leg of mutton ...	Sheep	*B. enteritis* ...	Bacillus not isolated, but diagnosis from serum tests.
99	Griefswald ...	1904	July ...	54	—	Meat (beef) ...	Ox ...	*B. suipestifer*	No information as to disease of the animal.
100	Berlin	1904	July ...	7	1	Meat pudding ...	—	Gaertner ...	No information as to source of infection.
101	Holland ...	1904	July ...	—	1	Meat and internal organs.	Calf ...	Gaertner ...	Animal diseased.
102	Zurich... ...	1904	September	2 families	2	—	Fish ...	Gaertner ...	Fish several days in transit and only eaten 24, 36, and some 48 hours after cooking.
103	Leipzig ...	1905	May ...	200	2	Meat (eaten raw) ...	Cow...	*B. enteritidis*	Cow emergency slaughtered for udder disease.
104	Alsfeld ...	1905	June ...	8	0	Salt pork	Pig ...	*B. suipestifer*	Food harmless at first, but pathogenic after being kept for a day and then warmed up.
105	König	1905	August ...	22	1	"Puddingspeise" ...	—	Gaertner ...	—
106	Leipzig ...	1906	January...	250	0	Tinned beans ...	—	Gaertner ...	Tin only opened just before the meal.
107	Saarbrücken ...	1906	July ...	16	—	Roast pork... ...	Pig ...	*B. suipestifer*	—
108	— (Described by Abraham.)	1906	(Summer)	28	—	Pike...	Fish...	Gaertner ...	No clear information as to source of infection.
109	Berlin	1906	September	90	2	Chopped beef ...	Ox ...	*B. suipestifer*	No information available as to the health of the animal.

110	— (Described by Fromme.)	1906	October...	32	0	Chopped ham (raw)	Pig ...	*B. suipestifer*	Upon the otherwise sound animal the butcher found an abscess upon one cheek, which was removed after the death of the animal. Later he noticed, in cutting up the pig, an abscess which he removed in the slaughter-house. In the slaughter-house list the pig was described as sound.
111	Frankfurt ...	1907	July ...	54	—	Sausage	—	*B. suipestifer*	—
112	Halle	1907	August ...	68	0	Chopped meat and Sülze.	—	*B. suipestifer*	No mention that the animals were diseased.
113	Rätzlingen ...	1907	December	21	2	Sülze	Cow...	*B. suipestifer*	Cow emergency slaughtered on account of gastro-enteritis.
114	Rostock ...	1907	Nov.–Dec.	73	—	Liver and lung sausage.	Pig ...	*B. enteritidis*	In the lung of one of the pigs was a diseased patch which was removed before the lungs were used for sausages. This may possibly be related, but no definite evidence.
115	Flanders ...	1907	—	58	1	Meat	Horse	*B. suipestifer*	The horse suffered from gastro-enteritis and had an epigastric abscess.
116	Switzerland ...	1907	—	36	4	Liver sausages ...	Pig ...	Gaertner ...	No information as to method of infection.
117	Frankfurt ...	1908	May ...	43	—	Blood sausage ...	Pig ...	*B. suipestifer*	No information as to method of infection.
118	Leipzig ...	1908	August ...	24	0	Ham	Pig ...	*B. suipestifer*	—
119	Magdeburg ...	1908	August ...	200+	—	Meat	Pigand Ox.	*B. suipestifer*	—
120	Altkloster ...	1908	October...	68	—	Chopped meat (raw)	Horse.	*B. suipestifer*	Animal apparently healthy.
121	Metz	1909	April ...	247	—	Meat and "Schwartenmagen."	Pig ...	*B. enteritidis*	No information as to the health of the animal.
122	St. Johann ...	1909	July ...	97	—	Meat	Ox ...	*B. enteriditis*	Animal emergency slaughtered on account of rupture of the bladder from stone.
123	H. (Zurich Canton).	1910	May ...	49	1	Salted pork (11 cases) "Schwartenmagen" (38 cases).	Pig ... Ox ...	Gaertner ...	—

ADDENDUM B.

Circular.

Sanitary Authorities,

England and Wales.

Outbreaks of Food-poisoning.

Local Government Board,
Whitehall, S.W.
12th September, 1911.

Sir,

I am directed by the Local Government Board to forward, for the information of the Council, the enclosed copies of a Memorandum which they have caused to be prepared, on the procedure which should be followed in dealing with outbreaks of illness suspected of having been due to the consumption of certain foods. I am to request that you will hand one copy of the Memorandum to the Medical Officer of Health for his information.

The Memorandum will be placed on sale so that copies may shortly be obtained from Messrs. Wyman & Sons, Ltd., Fetter Lane, E.C., either directly or through any bookseller.

I am, Sir,
Your obedient Servant,
H. C. MONRO.
Secretary.

The Town Clerk.
or
The Clerk to the District Council.

Memorandum by the Local Government Board on the Investigation of Outbreaks of Illness suspected to be due to Food-poisoning.

From time to time information of occurrence of cases of illness attributed to food-poisoning reaches the Local Government Board from medical officers of health, coroners, medical practitioners and others, or is obtained from newspaper reports. Occasionally the cases in question result from contamination of foods by inorganic poisons (*e.g.*, arsenic and lead), more frequently and especially in the case of meat foods, the poisoning has been of bacterial origin.*

The information obtained in this way as to the occurrence of serious cases or outbreaks of food-poisoning in England and Wales is by no means complete, and in many instances the information has reached the Board at a stage too late for any satisfactory investigation to be made by their Inspectors, or any useful assistance given to the officers of local authorities in dealing with the matter. It is anticipated that if more complete and timely information as to suspected food-poisoning were to reach the Board, it might sometimes be possible for them to secure the adoption of

* In the present Memorandum, the term "food-poisoning" is intended to include both classes of cases, but does not include instances in which foods, such as milk, ice-cream or shell fish, have produced any of the notifiable infectious diseases for example, scarlet fever or enteric fever.

effective preventive measures, while a better opportunity would be afforded for elucidating points in the causation of food-poisoning which at present are obscure.

Where a medical officer of health becomes aware of the occurrence of a considerable number of cases of food poisoning in his district (for example, resulting from the contamination of particular preparations of meat distributed locally on a large scale), he should report it forthwith to the Board (*see* Article XIX (15) of the Board's General Order of 13th December, 1910, and Article 18 (16) of the Sanitary Officers (London) Order, 1891), and he is, moreover, required by Article XIX (16) of the Order to furnish to the Board a copy of any special report or reports which he makes to his authority on the results of his inquiries into the outbreak.

The case is different where a medical officer of health learns that only a few persons in his district have been attacked by food-poisoning—for example, one or two members of a household suffering in consequence of the consumption of canned food from a particular tin. Although this does not constitute a serious outbreak of disease in the district, other canned foods of the same origin may be causing illness elsewhere. Instances have lately come to the Board's knowledge in which the collection of facts as to scattered cases occurring in several sanitary districts has had significant results, and they would accordingly be glad of prompt information whenever such cases come to the notice of the medical officer of health.

In any outbreak where the implicated food has been prepared in the district, it is advisable that as soon as the medical officer of health has established the probability that a particular food is at fault, he should at once proceed to make detailed investigations into the conditions of its preparation and obtain material for chemical or bacteriological examination. The complete history of all the cases attacked can be obtained simultaneously through assistants or by inquiry made subsequently. The determination of the circumstances in which food-poisoning has occurred often turns upon the elucidation of apparently trivial points, and after some days' interval it is impossible to rely on accurate recollection of them. Moreover, as time goes on inaccurate statements get repeated and believed, and it is difficult to get at the facts. It is neither desirable nor necessary to await the result of chemical or bacteriological examinations before commencing inquiries calculated to throw light on the manner in which the poisonous elements (bacterial or other) gained access to the food. Supplementary inquiries can always be made as a result of laboratory findings.

For convenience of reference, a list of headings for inquiry in the case of outbreaks of poisoning attributed to meat foods is appended to this Memorandum.

Where the food suspected to have caused poisoning has not been prepared in the district it is important to secure the co-operation of the vendor, who should be invited to produce original packages, invoices, and any other facts available to show by what manufacturing or distributing firm the implicated food was supplied to him, and on what date and in what bulk.

The Board would be glad to be informed of the facts obtained in any such cases.

Collection and Examination of Material.

It is important to secure samples of any remaining portions of the actual food which has been consumed by persons attacked, as well as samples of food of similar origin, or food prepared from the same ingredients. Perishable articles should at once be placed in an ice box or cold store pending examination which should be commenced as early as possible. In the case of canned goods, the cans with the labels intact should be preserved. Unopened cans, corresponding in all particulars with those suspected, should also be obtained if possible.

Chemical Examination.—Where the circumstances point to poisoning not of bacterial origin, samples with all the information available should forthwith be sent for chemical examination, and this would in ordinary circumstances be made by the public analyst of the district.

In cases of meat food-poisoning samples are sometimes sent to the analyst to be examined for the presence of "ptomaines." Ordinarily little is to be gained by so doing. It is by no means certain that "ptomaines" in the sense of alkaloidal substances produced by bacterial action are present in meat foods which have caused poisoning, and the significance of the reactions which are held to demonstrate the presence of these substances is a matter of considerable doubt. In such cases, however, chemical analyses may be valuable for the determination of special points, such as the presence or absence of preservatives and their nature, the determination of acidity, saltness, and like matters.

Bacteriological Examination.—It is of obvious advantage in the investigation of cases of food-poisoning that arrangements for any necessary bacteriological examination should have been considered beforehand and the delay and trouble of making special emergency arrangements avoided.

Where samples are liable to be delayed in transit it is desirable to send them in receptacles which are surrounded by ice or otherwise insulated.

When it appears desirable to procure for bacteriological examination material from persons who have been attacked by food-poisoning, the advice of the bacteriologist should be obtained in order that the material may be transmitted with proper precautions and in a condition suitable for examination.

It is important that material should be available for any investigations which the Board may desire to make through their own officers. Where such an investigation is directed an early intimation will be sent to the medical officer of health. In all cases, however, it is desirable that the chemist or bacteriologist consulted should be requested to preserve samples under suitable conditions until it has been ascertained that there is no further use for them.

The Board are aware that neither medical practitioners nor the general public are under statutory obligation to report cases of food-poisoning to the medical officer of health. It is believed, however, that if it is generally understood in a district that the local authority, with a view to safeguarding the public health, is pre-

pared to authorise any necessary inquiries and investigations, the arrangements made will receive general approval and tend to secure the co-operation of all concerned.

Local Government Board,
September, 1911.

APPENDIX.

HEADINGS OF INQUIRY INTO OUTBREAKS OF POISONING BY MEAT FOODS.

What cases heard of; steps taken to secure complete list of cases, *e.g.*, inquiries of medical practitioners, neighbouring medical officers of health and others.

Evidence implicating particular food or foods as cause of outbreak.

Evidence implicating any particular ingredient of the food.

Origin of suspected food or ingredient.

Inquiries in affected households.

Names and ages of persons (*a*) in each household, (*b*) those ill, (*c*) those partaking of suspected food.

Persons affected—(*a*) slightly, (*b*) seriously, (*c*) fatally; with date and hour of partaking of food in each case and date and hour of first symptoms in each case.

Clinical character of illness.

Particular food implicated. Date of purchase, source; any form of domestic preparation applied (*e.g.*, cooking); if so, how long and to what degree; if canned meat, when opened, &c.

Inquiries at place of preparation (when food implicated has been prepared in the district).

Address or description of place of preparation; name and address of owner or occupier; number of workers employed (male and female); nature of employment in each case. Any engaged also in other employments which might have connection with contamination of the suspected food.

Meat concerned; its nature, where obtained, amount used on days concerned, how and where stored.

Evidence, positive or negative, of unsoundness of the meat.

Evidence, positive or negative, as to disease of animal during life or ascertained post-mortem.

Possibilities of infection at slaughterhouse or place of storage.

Sanitary condition of bakehouse or food preparing place (including distance from possible sources of contamination, *e.g.*, middenstead, ash-pit, privy, W.C.; slaughterhouse, stable); positions of drain openings; ventilation; general sanitary condition.

Cleanliness of tables, floors, vessels, utensils, &c.

Preparation of food (exact details of methods employed, including history and condition of various component parts besides the meat, *e.g.*, pastry, stock and jelly for pork pies, skins of sausages, &c.).

Handling of the food (possible contamination by "carrier" of bacteria associated with food poisoning, (*a*) before cooking, (*b*) during cooking, (*c*) after cooking, *e.g.*, transfer into moulds, &c.).

Temperature reached in cooking; any experimental verification of temperature, especially as regards interior of mass; any reason to suspect under-cooking of whole or part.

Cooling. Where food placed during cooling. Possible opportunities of contamination.

Health of workers previous to outbreak, especially in regard to diarrhœa; their habits as to cleanliness. What W.C. accommodation for workers (where situated and condition). Arrangements for washing hands and their use.

Collection and examination of Material for bacteriological examination.

Samples collected (dates, description and quantities) of:—

Food materials. (*a*) Portions left over by patients, (*b*) obtained at shops, stores or places of preparation.

Clinical materials. (*a*) Blood from patients or suspected "carriers," (*b*) post-mortem specimens.

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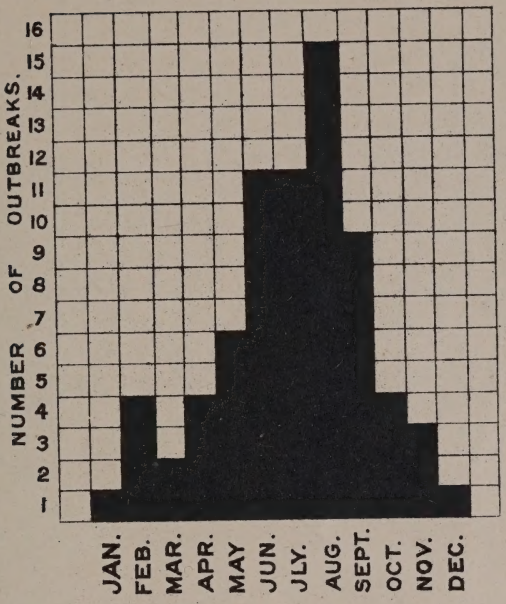
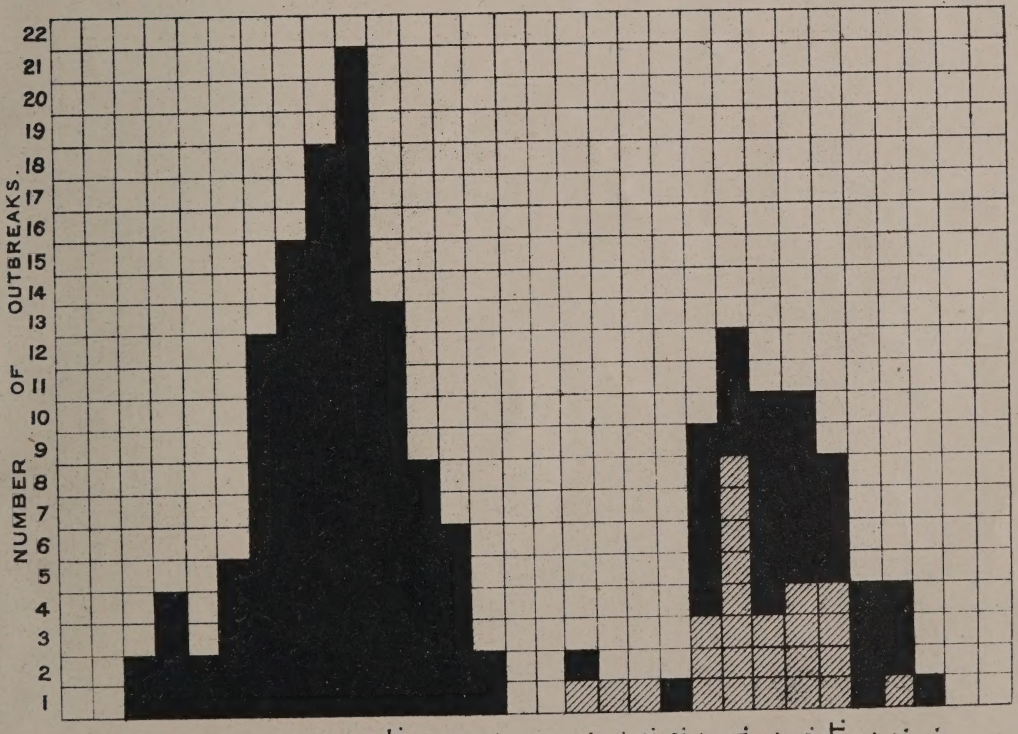
Chart 1.

71 BRITISH OUTBREAKS.





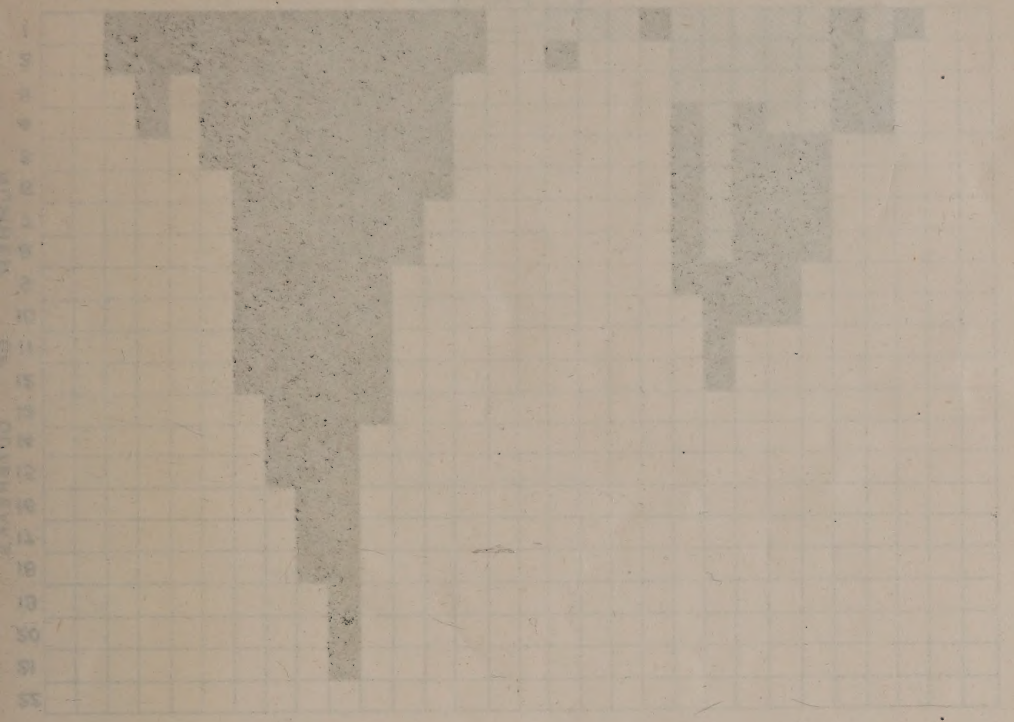
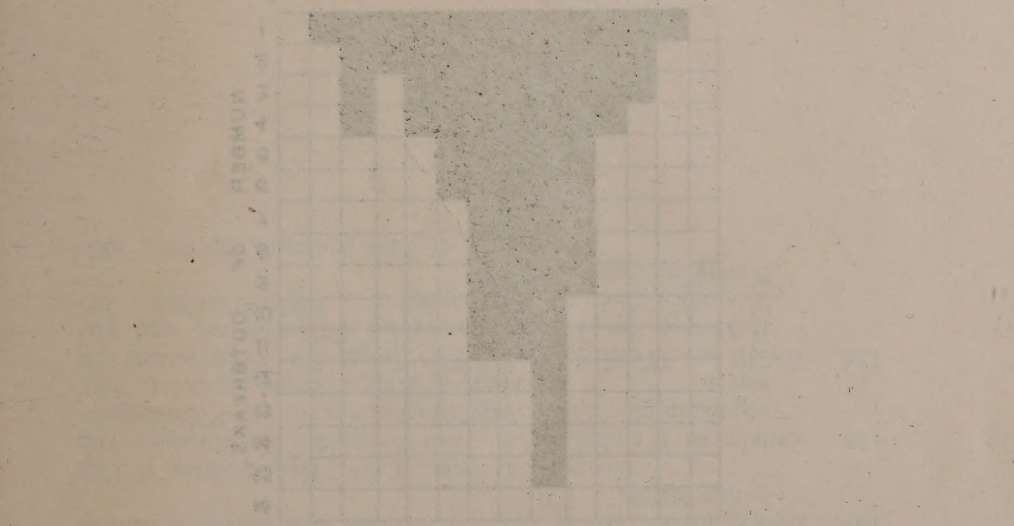
BRITISH AND CONTINENTAL OUTBREAKS.

Chart 2.

OUTBREAKS ASCRIBED TO GAERTNER GROUP BACILLI.

Chart 3.

▨ *British.* ■ *Continental.*

1650.11798/99. 2000.8.13.

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